PHILIP MORRIS
SUBMISSION
ON
OSHA'S PROPOSED RULE ON
INDOOR AIR QUALITY

(59 FR 15968)

TEXT

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INTRODUCTION

INTRODUCTION

On September 20, 1991, OSHA issued a Request for Information on Occupational Exposure to Indoor Air Pollutants (56 FR 47892) (hereinafter "RFI") in an effort to obtain the necessary information to determine whether regulatory action regarding indoor air quality (hereinafter "IAQ") was appropriate. (56 FR 47892). The RFI sought information relating to the health effects attributable to poor IAQ, ventilation system performance, exposure assessments and abatement methods. Over 1,200 interested parties commented to OSHA in response to the RFI.

On April 5, 1994, OSHA published a Notice of Proposed Rulemaking on Indoor Air Quality (hereinafter "NPR"). (59 FR 15968) The NPR sets out the basis for OSHA's proposal to regulate IAQ as set forth therein, including occupational exposure to environmental tobacco smoke (hereinafter "ETS").

This submission sets forth and documents the positions of Philip Morris on the issues raised by the Notice of Proposed Rulemaking. These comments, which supplement the Philip Morris submission to the RFI docket, address issues including the scientific, legal, and practical aspects of OSHA's proposed rule. In general:

SECTION II

LEGAL ANALYSIS OF THE PROPOSED RULE

LEGAL ANALYSIS OF THE PROPOSED RULE

I. OSHA HAS EXCEEDED ITS LIMITED STANDARD-SETTING AUTHORITY IN THE PROPOSED STANDARD

A. OSHA's standard-setting authority is limited by the Occupational Safety and Health Act

In §§ 3(8) and 6(b)(5) of the Occupational Safety and Health Act of 1970 (29 USC § 651 et seq.) (hereinafter, "OSH Act" or "Act"), Congress established OSHA's standard-setting authority:

§ 3(8)

> 'Standard' means the adoption or use of one or more practices, means, methods, operations, or processes, reasonably necessary or appropriate to provide safe or healthful employment and places of employment.[1/]

To develop occupational safety and health standards about toxic materials or harmful physical agents, the Secretary of Labor shall

§ 6(b)(5)

> set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life.[2/]

1/ 29 U.S.C. § 652(8) [emphasis added].

2/ 29 U.S.C. § 655(b)(5) [emphasis added].

The limitations that these provisions place upon OSHA's standard-setting authority were first interpreted by the United States Supreme Court in Industrial Union Department, AFL-CIO v. American Petroleum Institute, 448 U.S. 607, 65 L. Ed.2d 1010, 100 S. Ct. 2844 (1980) (hereinafter "Benzene").

B. The U.S. Supreme Court's interpretation of §§ 3(8) and 6(b)(5) of the OSH Act

In Benzene, a Supreme Court plurality held that in regulating toxic materials or harmful physical agents, OSHA's authority to promulgate such standards is governed by §§ 3(8) and 6(b)(5) of the OSH Act. As Justice Powell explained in his concurring opinion:

> . . .[Sections] 6(b)(5) and 3(8) of the [Act] must be read together. They require OSHA to make a threshold finding that proposed occupational health standards are reasonably necessary to provide safe workplaces. When OSHA acts to [limit exposure to a substance], therefore, it must find that (i) [existing] permissible exposure levels create a significant risk of material health impairment; and (ii) a reduction of those levels would significantly reduce the hazard.

(Benzene, 448 U.S. at 664-665) [emphasis added]

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Thus, as a threshold matter, to promulgate an occupational health standard, OSHA must initially follow a "two-pronged" test by: (1) reaching a threshold finding that the proposed standard is reasonably necessary to provide a safe workplace, by establishing that the existing workplace exposure to

the substance in question creates a significant risk of material health impairment; and (2) establishing that the standard's requirements, including the level of exposure permitted by the standard, are reasonably necessary to significantly reduce that significant risk.

Although the Benzene decision was a plurality decision, its holding was adopted by a majority of the Supreme Court in American Textile Manufacturer's Institute v. Donovan, 452 U.S. 490 ("Cotton Dust"), and by every federal court of appeals that has since addressed a challenge to a newly-promulgated OSHA standard.[3/] Federal courts of appeals have also followed the Benzene decision for the proposition that an employer whose practices do not pose a

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[3/] See, e.g., AFL-CIO v. OSHA, 65 F.2d 962, 972-73 (11th Cir. 1992) (air contaminants); American Dental Association v. Martin, 984 F.2d 823, 827, 832 (7th Cir. 1992) (bloodborne pathogens); International Union, UAW v. OSHA, 938 F.2d 1310, 1316 (D.C. Cir. 1991) (lockout/tagout); National Grain and Feed Association, Inc. v. OSHA, 866 F.2d 717, 737 (5th Cir. 1989) (grain handling); International Union UAW v. Pendergrass, 878 F.2d 389, 392 (D.C. Cir. 1989) (formaldehyde); Building & Construction Trades Dept., AFL-CIO v. Brock, 838 F.2d 1258, 1263 (D.C. Cir. 1988) (asbestos); Associated Builders and Contractors, Inc. v. Brock, 862 F.2d 63, 67 (3rd Cir. 1988) (hazard communication); Public Citizen Health Research Group v. Tyson, 796 F.2d 1479, 1496 (D.C. Cir. 1986) (ethylene oxide); Forging Industry Association v. Sec. of Labor, 773 F.2d 1436 (4th Cir. 1985) (noise); ASARCO, Inc. v. OSHA, 746 F.2d 483, 490 (9th Cir. 1984) (arsenic); United Steelworkers of America, etc. v. Marshall, 647 F.2d 1189, 1245 (D.C. Cir. 1980) (lead); Texas Indep. Ginners' Ass'n v. Marshall, 630 F.2d 398, 407 (5th Cir. 1980) (cotton ginning). See also National Cottonseed Products Association v. Brock, 825 F.2d 482, 484 (D.C. Cir. 1987) (medical surveillance of cotton dust exposure); Public Citizen Health Research Group, et al. v. Auchter, 702 F.2d 1150, 1156 (D.C. Cir. 1983) (emergency temporary standard on ethylene oxide); Louisiana Chemical Association v. Bingham, 657 F.2d 777, 782 (5th Cir. 1981) (access to exposure and medical records).

significant risk of material health impairment cannot be held in violation of an OSHA safety and health standard. (See, for example, Pratt & Whitney Aircraft, et al. v. Secretary of Labor, 715 F.2d 57 (2d Cir. 1983)) Nine (9) different federal courts of appeals follow the significant risk test articulated by the plurality in the Benzene decision.[4/]

In Benzene, the Supreme Court prohibited OSHA from applying its then-standard policy on carcinogens to regulate benzene. The OSHA policy stated that "in the absence of definitive proof of a safe level [of a substance to which employees are exposed], it must be assumed that any level above zero presents some increased risk of cancer." (448 U.S. at 635-36) Pursuant to this policy, OSHA contended that it was justified in regulating carcinogens to the lowest feasible level, which, in some cases, meant a total prohibition of the substance at issue. The Supreme Court disagreed, holding that OSHA could not rely on the unsupported assumption that any level above zero of a given substance would pose a significant risk. (448 U.S. at 653) According to the Court, before OSHA could ban a substance

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[4/]See, e.g., Donovan v. General Motors Corp., 764 F.2d 32, 36 (1st Cir. 1985); Pratt and Whitney Aircraft v. Donovan , 715 F.2d 57, 61-62 (2d Cir. 1983); Pratt and Whitney Aircraft v. Secretary of Labor, 649 F.2d 96, 103-104 (2d Cir. 1981). See Kelly Springfield Tire Co., Inc. v. Donovan, 79 F.2d 317, 323 (5th Cir. 1984); Modern Drop Forge Co. v. Secretary of Labor, 683 F.2d 1105, 1115 (7th Cir. 1982). Super Excavators v. Occupational Safety and Health Review Commission, 674 F.2d 592, 595 (7th Cir. 1981). Two of these cases were decided by circuits that had not yet addressed a challenge to a newly-promulgated standard.

completely from the workplace, it had to demonstrate that eliminating "any level" of the substance, "no matter how minute the exposure," was necessary to yield a significant "discernable benefit." (448 U.S. at 645)

In direct contravention of the U.S. Supreme Court's ruling in Benzene, OSHA now proposes to ban environmental tobacco smoke (ETS) completely from all areas of an enclosed workplace where employees perform work. The Agency proposes this drastic course even though it admittedly lacks any data to suggest that the elimination of "any level" of ETS, "no matter how minute the exposure," is necessary to yield a significant "discernible benefit." Exactly like OSHA's attempted regulation of benzene over a decade ago, the Agency is now relying on the unsupported assumption that any level of ETS above zero poses an increased risk of material health impairment in order to justify a regulation that will, in effect, reduce exposure for nonsmoking employees to zero.

II. OSHA HAS NOT COMPLIED WITH THE TWO-PRONGED SIGNIFICANT RISK TEST ARTICULATED IN BENZENE

A. OSHA's assessment of the risk allegedly posed by existing workplace levels of ETS is fatally flawed

As the Supreme Court explained in Benzene, while OSHA has "no duty to calculate the exact probability of harm, it does have an obligation to find that a significant risk is present before it can characterize a place of employment as 'unsafe.'" (448 U.S. at 655) In AFL-CIO v. OSHA, 965 F.2d 962 (11th Cir. 1992) (Air Contaminants), the Eleventh Circuit discussed the quantification of risk, explaining that "OSHA must provide at least an estimate of the actual [workplace] risk associated with a particular toxic substance." The Court found that "OSHA has satisfied this requirement by estimating either the number of workers likely to suffer the effects of exposure or the percentage of risk to any particular worker." (965 F.2d at 973)

In Benzene, the Supreme Court set forth the quantitative parameters within which a significant risk lies. The Court stated: "If, for example, . . . the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal, a reasonable person might well consider the risk significant and take appropriate steps to decrease or eliminate it." (448 U.S. at 655) [emphasis added]

Following this example, OSHA has embraced the 1 in 1,000 test as a policy guideline, consistently promulgating occupational safety and health standards where it estimates the level of risk from existing workplace exposure to be one excess death per 1,000 employees.[5/] This means that compared to the risk faced by the general population from exposure away from the workplace, an additional one person out of every one thousand will suffer material health impairment because of exposure at work. Additionally, the federal courts have recognized this policy as OSHA's regulatory guideline. (See, AFL-CIO v. OSHA, 965 F.2d. 962, 973 n. 15 (11th Cir. 1992); International Union, UAW v. Pendergrass, 878 F.2d 389, 392 (D.C. Cir. 1989).)

OSHA has estimated the risk of lung cancer and heart disease allegedly posed by workplace exposure to ETS. According to OSHA's estimate, the lifetime occupational risk of lung cancer in nonsmoking employees from exposure to ETS at the workplace is 0.4 to 1 case per 1,000 exposed employees. OSHA estimates that the lifetime occupational risk of heart disease in nonsmoking employees

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[5/] See, e.g., 57 FR 42102, 42206 (Occupational Exposure to Cadmium); 56 FR 64004, 64037 (Bloodborne Pathogens); 55 FR 32736, 32786 (Occupational Exposure to Butadiene); 54 FR 9294, 9312 (Hazardous Waste Operations and Emergency Response); 54 FR 2332, 2675 (Occupational Exposure to Air Contaminants - Acrylamide); Id. at 2678 (Amitrole); Id. at 2680 (Carbon Tetrachloride); Id. at 2682 (Chloroform); Id. at 2691 (P-Toluidine); Id. at 2694 (Vinyl Bromide); 52 FR 34460, 34507 (Occupational Exposure to Benzene); 51 FR 22612, 22646-7 (Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite); 49 FR 25734, 25764 (Occupational Exposure to Ethylene Oxide); 48 FR 1864 (Occupational Exposure to Inorganic Arsenic).

from workplace exposure to ETS is 7 to 16 cases per 1,000 exposed employees.

In developing these risk estimates, OSHA uncritically relies upon one epidemiologic study to determine the alleged risk of lung cancer due to ETS exposure in the workplace, without adequate discussion of the study's quality or weaknesses. (59 FR 15995) The workplace data from 13 other available studies on nonsmoker lung cancer, eight of which were conducted in the U.S., were ignored. OSHA also utilizes a single epidemiologic study on cardiovascular disease (CVD) that addresses spousal smoking in the home to estimate the risk allegedly due to ETS exposure in the workplace, while ignoring available data on reported ETS exposures and CVD in the workplace. (59 FR 15995)

In these two epidemiologic studies on which OSHA's risk estimates are based, ETS exposures were not measured directly. Instead, ETS exposures were estimated in the two studies by individual recollection of exposure. Accurate quantification of exposure cannot be ascertained through this kind of study. **No measured ambient exposure data for ETS are included in OSHA's estimate of the risk allegedly posed by ETS in the workplace.** Thus, OSHA offers an analysis of risk and a proposal for the complete elimination of ETS from indoor work areas **without reference to any measured exposure data.**

Despite OSHA's failure to consider such studies, measured exposure data for constituents of ETS in the air of indoor work environments are currently available. Indeed, OSHA's NPR does not reference a single ambient air monitoring study for the workplace published after 1991, the same year in which OSHA admitted that it had no adequate data on "current levels of exposure" for ETS. (59 FR 15990)

Although the studies were available, the OSHA NPR nevertheless states that "estimating the risk from exposure to ETS requires the use of some measure of the extent of exposure." (59 FR 15997) OSHA acknowledges failure to integrate measured ambient ETS exposure data into its analysis of significant risk and states that "[s]ince there is no definition of, nor an established method for quantifying, exposure, it is not possible to determine a 'dose limit' that would eliminate significant risk." (59 FR 16001) As discussed below, and demonstrated in the scientific portion of Philip Morris' comments, current exposure data on ETS levels in the workplace do not support the conclusion that ETS poses a significant risk of material health impairment to nonsmoking employees.

There are well over 100 published studies on ETS measurements in the air of public places, workplaces, restaurants and other locations, all of which were submitted by Philip Morris to OSHA in response to the 1991 Request for Information on

Occupational Exposure to Indoor Air Pollutants ("RFI"). (Ex. 3-1074) These studies report measurements for constituents of ETS in the air. The most commonly measured constituents are carbon monoxide, nicotine, and respirable suspended particles (RSP). The data from these numerous studies suggest that, overall, nonsmoker exposure to ETS under normal everyday conditions is actually very low.

For example, researchers report that there is very little difference in ambient levels of carbon monoxide in smoking and nonsmoking areas of workplaces and public places, and in homes with and without smokers.[6/] Other studies indicate that ETS contributes 10 to 50 percent of the total particles in the air of a typical public place in which smoking is permitted.[7/] Recent workplace air

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[6/]See Kirk, P., et al., "Environmental Tobacco Smoke in Indoor Air," in: Indoor and Ambient Air Quality. R. Perry and P. Kirk (Eds.). London, Selper Ltd., 99-112, 1988; Duncan, D. and Greavey, P., "Passive Smoking and Uptake of Carbon Monoxide in Flight Attendants" JAMA 251(20): 120-21, 1984; Cox, B. and Whichelow, M., "Carbon Monoxide Levels in the Breath of Smokers and Nonsmokers: Effect of Domestic Heating Systems," J Epidemiol Community Health 39: 75-78, 1985; Girman, J. and Traynor, G., "Indoor Concentrations," JAPCA 33(2): 89, 1983; Yocom, J., "Indoor Concentrations," JAPCA 33(2): 89, 1983; and Nitta, H., et al., "Measurements of Indoor Carbon Monoxide Levels Using Passive Samplers in Korea" in: Indoor Air '90. The Fifth International Conference on Indoor Air Quality and Climate, Toronto, Canada, July 29-August 3, 77-82, 1990. Philip Morris submitted these studies to OSHA on March 18, 1992, in response to OSHA's RFI (Ex. 3-1074).

[7/]See Baker, R. and Proctor, C., "The Origins and Properties of Environmental Tobacco Smoke," Env. Int. (16): 231-245, 1990; Guerin, M., et al., The Chemistry of Environmental Tobacco Smoke: Composition and Measurement, (Lewis Publishers: Chelsea, Michigan, 1992); Reasor, M. and Will, J., "Assessing Exposure to Environmental Tobacco Smoke: Is It Valid to Extrapolate from

(continued...)

monitoring studies report that levels of respirable suspended particles (RSP) range from about 5.8 to 45.9 ug/m^3, averaging only 21.3 ug/m^3. (See Table 5 attached at Part IV.)

2/(...continued)
Active Smoke?" Journal of Smoking Related Diseases 2(1): 111-127, 1991; Proctor, C. and Dymond, H., "The Measurement of ETS Through Absorption/Desorption Procedures" in: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 82-89, 1990; Nystrom, C., et al., "Assessing the Impact of Environmental Tobacco Smoke on Indoor Air Quality: Current Status" in: Proceedings of the ASHRAE Conference, IAQ '86. April 20-23, 1986, Atlanta, Georgia, 213-233, 1986; Rawbone, R., "The Aging of Sidestream Tobacco Smoke Components in Ambient Environments" in: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 55-61, 1990; Piade, J., et al., "Assessment of ETS Impact on Office Air Quality" in: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 112-119, 1990; Scherer, G., et al., "Importance of Exposure to Gaseous and Particulate Phase Components of Tobacco Smoke in Active and Passive Smokers," Occup Env Health (62): 459-466, 1990; Rodgman, A., "Environmental Tobacco Smoke," Reg Tox and Pharm 16:223-244, 1992; Kirk, P., et al., "Environmental Tobacco Smoke in Indoor Air" in: Indoor and Ambient Air Quality. R. Perry and P. Kirk (Eds.). London, Selper Ltd., 99-112, 1988; Carson, J. and Erikson, C., "Results from a Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988; Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," Journal of Occupational Medicine 26(1): 57-62, 1987; Sterling T. and Mueller, B., "Concentrations of Nicotine, RSP, CO and CO_2 in Nonsmoking Areas of Offices Ventilated by Air Recirculated From Smoking Designated Areas," Am Ind Hyg Assoc J 49(9): 423-426, 1988; Holcomb, L., "Indoor Air Quality and Environmental Tobacco Smoke: Concentration and Exposure," Environment Int (19):9-40, 1993; Oldaker, G., et al., "Results From Surveys of Environmental Tobacco Smoke in Restaurants" in: Indoor Air Quality. H. Kasuga (Ed.). Springer-Verlag, Berlin, Heidelberg, 99-104, 1990; Turner, S., "The Measurement of Environmental Tobacco Smoke in 585 Office Environments," Environment Int (18): 19-28, 1992; Proctor, C., et al., "Measurements of Environmental Tobacco Smoke in an Air-Conditioned Office Building," Environ Technol Letters (10): 1003- 1018, 1989; and Proctor, C., et al., "Measurements of Environmental Tobacco Smoke in an Air-Conditioned Office Building" in: Present and Future of Indoor Air Quality. C. J. Bieva, et al. (eds.). Brussels, Elsevier, 169-172, 1989. Philip Morris submitted these studies to OSHA on March 18, 1992, in response to OSHA's RFI (Ex. 3-1074).

Also, recent workplace air monitoring studies report that actual measured nicotine levels range from about 0.17 to 7.2 ug/m^3, averaging 2.8 ug/m^3. (See Table 5 attached at Part IV.) In terms of cigarette equivalents, typical measurements of nicotine range from an exposure equivalent of 1/100 to less than 1/1,000 of one filter cigarette per hour. This means that a nonsmoker would have to spend from 100 to 1,000 hours or more in an office, restaurant or public place where smoking is unrestricted, in order to be exposed to the nicotine equivalent of a single cigarette.[8/]

[8/] See, Carson, J. and Erikson, C., "Results from a Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988; Oldaker, G., et al., "Results From Surveys of Environmental Tobacco Smoke in Offices and Restaurants" in: Indoor Air Quality. H. Kasuga (Ed.). Springer-Verlag, Berlin, Heidelberg, 99-104, 1990; Hinds, W. and First, M., "Concentrations of Nicotine and Tobacco Smoke in Public Places," New England Journal of Medicine 292(16): 844-845, 1975; Badre, R., et al., "Pollution Atmospherique par la Fumee de Tabac (Atmospheric Pollution by Smoking)," Ann Pharm Fr 36(9-10): 443-452, 1978. Translation; Jenkins, R., et al., "Development and Application of a Thermal Desorption-Based Method for the Determination of Nicotine in Indoor Environments" in: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 557-566, 1988; Muramatsu, J., et al., "Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor," Environ Res 35: 218-227, 1984; Muramatsu, J., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch Occup Environ Health 59: 545-550, 1987; Thompson, C., et al., "A Thermal Desorption Method for the Determination of Nicotine in Indoor Environments," Envir Sci Tech 23: 429-435, 1989; Foliart, D., et al., "Passive Absorption of Nicotine in Airline Flight Attendants," New England Journal of Medicine 308(18): 1105, 1983; and Oldaker, G., and Conrad, F., "Estimation of the Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Envir Sci Tech 21: 994-999, 1987. Philip Morris submitted these studies to OSHA on March 18, 1992, in response to OSHA's RFI (Ex. 3-1074).

These data on minimal workplace exposure were confirmed recently by Mr. Simon Turner of Healthy Buildings International, who testified before the Maryland Occupational Safety and Health Advisory Board on December 16, 1993, regarding that state's proposed workplace smoking regulation. (Transcript, Vol. II, page 159, lines 6-10) He testified that, according to the published studies, "the [ETS] levels that are actually out there are between 14 and 23 times lower than the ones that are claimed by Mr. [James] Repace [of the U.S. Environmental Protection Agency]."[9/] (Id. at page 160, lines 1-3.)

Additional data on workplace exposure were submitted to the Maryland Division of Labor and Industry at a May 3, 1994, public hearing on the same proposed regulation about which Mr. Turner testified. Dr. Domingo M. Aviado, a scientist, medical doctor, and preeminent expert in occupational toxicology and tobacco smoke constituents, testified that "levels of purported ETS constituents are unlikely even to approach workplace standards," such as the OSHA "permissible exposure limits" (PELs) and the sometimes significantly lower "threshold limit values" (TLVs) recommended by the American Conference of Governmental Industrial

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[9/]Mr. Repace's studies and models of the levels of certain ETS constituents in selected workplaces were relied upon by OSHA in the NPR. Mr. Repace also contributed to EPA's recent risk assessment on the alleged health effects of ETS exposure in the home (discussed below).

Hygienists.[10/] (Transcript, Vol. VI, page 116, lines 13-15) Dr. Aviado concluded: "There is insufficient biomedical basis to support the general proposition that ETS exposure causes occupational diseases." (Id. at page 124, lines 9-11) An accurate assessment by OSHA of the risk, if any, posed by existing unregulated levels of ETS in the workplace must thus conclude that any such actual exposure does not pose a significant risk of

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[10/]Dr. Aviado has practiced, taught, and researched in the field of occupational toxicology and medical pharmacology for over 45 years. On obtaining his medical degree in 1948 from the University of Pennsylvania, he began research and teaching at that university in medical pharmacology, progressing from instructor to professor of pharmacology from 1948 to 1977. (Transcript, Vol. VI, page 112, lines 12-17) At the University of Pennsylvania Medical School, Dr. Aviado's sponsored research was funded by the Department of Defense, Food and Drug Administration, Consumer Products Safety Commission, National Institutes of Health, Council for Tobacco Research, and the pharmaceutical industry. (Id. at page 112, lines 18-21; page 113, lines 1-5) In 1978, Dr. Aviado was appointed to the Environmental Protection Agency's Scientific Advisory Board (EPA-SAB) and was the physician member of the Clean Air Scientific Advisory Committee, mandated by the 1977 Clean Air Act. (Id. at page 115, lines 20-21; page 116, lines 1-3) Since 1978, he has been President of Atmospheric Health Sciences, which provides consulting services in the fields of pharmacological sciences, occupational toxicology and medicine. (Id. at page 113, lines 10-14)

Approximately half of Dr. Aviado's research activity has related to the alleged health effects of chemical constituents of tobacco smoke. He has published 300 articles, 10 monographs, one textbook of pharmacology and two medical dictionaries. (Id. at page 113, lines 14-18) Significantly, Dr. Aviado has recently authored a chapter entitled "Complex Mixtures of Tobacco Smoke and the Occupational Environment" in the 1993/94 edition of Patty's Industrial Hygiene & Toxicology. (Id. at page 114, lines 5-8) This chapter, along with Dr. Aviado's other chapter on fluorocarbons, was thoroughly reviewed both by the editors and several outside reviewers. (Id. at page 115, lines 6-8) For the past four decades, Patty's has been widely recognized as the authoritative reference on industrial hygiene and toxicology. (Id. at page 115, lines 8-11)

material health impairment. (See Building and Construction Trades Dept., AFL-CIO v. Brock, 838 F.2d 1258, 1266 (D.C. Cir. 1988))

At the same May 3, 1994, Maryland hearing, Dr. Roger Jenkins of Oak Ridge National Laboratory (ORNL) testified about his laboratory's ongoing study examining actual ETS exposures in the workplace. The ORNL study, described as "the largest ever conducted in the United States of the personal exposure of individuals to environmental tobacco smoke in the home and workplace," involves 1,600 randomly-selected employees in 16 U.S. cities who each wear a personal ETS exposure monitor at work, and a separate monitor while away from work. (Transcript, Vol. VI, page 87, lines 6-16) The two monitors measure the various ETS constituents to which the employee is exposed throughout a 24-hour period, eight hours at work and 16 hours away from work.[11/] (Id. at page 89, lines 8-13; page 104, lines 5-21; page 105, lines 1-11.) When questioned about the preliminary results of the ORNL study, Dr. Jenkins responded that data from the six cities where the study has been completed show that exposure to ETS constituents away from work is generally three to four times greater than

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[11/]The study measures actual exposure to the following ETS constituents: respirable suspended particulate (RSP) matter, UV absorbing particulate matter, fluorescing particulate matter, solanesol (a tobacco-specific terpene), nicotine, 3-ethenyl pyridine, and myosmine. (Transcript, Vol. VI, page 88, lines 6-12)

exposure at work.[12/] (Id. at page 93, lines 6-14.) Moreover, since exposure away from work is twice as long as exposure at work, total exposure away from work could be as much as six to eight times greater than workplace exposure. (Id. at page 93, lines 14-16.) According to Dr. Jenkins, if these trends hold for the rest of the 16 cities, then the ORNL data will support the position that ETS exposure away from work is "several factors greater" than workplace exposure. (Id. at page 92, lines 7-12.)

Dr. Jenkins pointed out that the preliminary results of the ORNL study are consistent with the results of a similar study conducted by Hazelton Laboratories in the United Kingdom. (Id. at page 91, lines 10-13.) Like the ORNL study, the Hazelton study involved the acquisition of 24-hour personal exposure samples for approximately 250 participants. (Id. at page 91, lines 13-16.) The Hazelton study reported that "workplace exposures to nicotine were about half those that occur in the home." (Id. at page 91, lines 19-20.)

The preliminary data from the ORNL study and the data from the Hazelton study are critical because they directly conflict with the recent EPA risk assessment on the alleged health effects of ETS (the "EPA Report," Ex. 8-311), which contended, without

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[12/]The "at work" environment is comprised of workplaces where there are "essentially no restrictions as to smoking." (Transcript, Vol. VI, page 95, line 21; page 96, lines 1-3; page 102, lines 6-8; page 102, lines 20-21; page 103, lines 1-4)

support, that workplace concentrations of ETS are "analogous" to concentrations to which spouses of smokers are exposed at home.[13/] (Id, at page 91, line 21; page 92, lines 1-4.) But given this unrebutted evidence of actual workplace exposure at existing levels, any reliance by OSHA on the EPA Report focusing on spousal exposure in the home to justify a workplace smoking regulation is unfounded.

As recently as March of this year, OSHA itself stated:

> Under the OSH Act, OSHA must make its own determinations of significant risk and feasibility and cannot, as ASH suggests, simply rely upon EPA's or any other agency's assessment of the evidence. That there is consensus among governmental agencies and scientific organizations concerning ETS's carcinogenicity does not obviate the need for OSHA to determine that occupational ETS exposure constitutes a significant risk. During the early stages of OSHA's consideration of this issue, it had to determine how to relate available assessments of risk to occupational settings.

ASH v. OSHA, No. 92-1661, Brief for the Secretary of Labor, Petition to Review a Final Decision of the Occupational Safety and Health Administration, page 32, n. 24 (D.C. Cir., March 1994) [emphasis added]

[13/]The EPA risk assessment, titled "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders," focuses almost exclusively on reported exposures to ETS from spousal smoking in the home, not workplace exposure.

In 1991, at oral argument in a related case, the OSHA representative stated:

> The principal risk assessments in this area are not definitive, because they rely heavily upon data developed in residential studies, rather than on actual occupational exposures. OSHA has noted that the conditions affecting occupational exposure vary widely, depending upon building size and type, ventilatory exchange rates, occupational density and other factors.

ASH v. OSHA, No. 89-1656, Transcript of Proceedings, page 12, lines 13-19, (D.C. Cir., May 6, 1991) [emphasis added] The OSHA representative then pointed out that

> the Surgeon General's report, on which ASH places heavy reliance, is typical of this group of studies, in that it expressly recognizes that the degree of risk due to exposure to ambient tobacco smoke is uncertain, and that additional and more accurate estimates [of] exposures in the workplace, in the home and in public places is necessary in this arena.

Id. at page 16, lines 10-16 [emphasis added]

In addition to its failure to consider all the workplace data, OSHA failed to consider the effects of the indoor air quality provisions of the proposed standard on its ETS risk assessment. For example, as discussed in detail below, improving the operation of a building's ventilation system will dilute ETS constituents significantly.

1. **The best available evidence related to existing levels of workplace ETS does not support a significant risk of material health impairment**

Section 6(b)(5) of the OSH Act requires any standard promulgated by OSHA to be based, to the extent feasible, on the "best available evidence." In Benzene, the Supreme Court pointed out that the Fifth Circuit below had held that OSHA's ban on dermal contact with benzene was not based on the "best available evidence." The Supreme Court stated:

> In light of §6(b)(5), which requires the Agency to promulgate standards on the basis of the 'best available evidence' and the 'latest available scientific data in the field,' the court held that where there is uncontradicted testimony that a simple test will resolve the issue, the Agency is required to acquire that information before 'promulgating regulations which would require an established industry to change long-followed work processes that are not demonstrably unsafe.' (581 F.2d, at 508)

(448 U.S. at 661)

The Supreme Court did not reach the Fifth Circuit's holding, finding that OSHA failed to make the required threshold finding that the dermal contact ban is "reasonably necessary or appropriate" to remove a significant risk of harm from such contact. (Id. at 662.)

Similarly, in Texas Independent Ginners Association v. Marshall, 630 F.2d, 398 (5th Cir. 1980), petitioners sought review

of an OSHA standard on cotton dust exposure in the cotton gin industry. In promulgating the standard, OSHA relied exclusively on studies demonstrating that exposure to cotton dust in the American textile industry and in foreign ginning operations caused byssinosis. Petitioners alleged that these studies were not the best available evidence, since a study which actually analyzed exposure to cotton dust in the American cotton gin industry was available. That study found no evidence of byssinosis or other chronic respiratory disease among American gin workers. The Fifth Circuit vacated the standard, holding in part that OSHA failed to base the regulation on the "best available evidence," as required by the OSH Act. The Court stated: "on further consideration, OSHA's continued refusal to consider this latest available scientific test would violate its statutory obligation to consider the best available evidence." (Id. at 413, fn. 48.)

a. Workplace exposure levels

Philip Morris submits that both the Hazelton study and the ORNL study discussed above are among the best data available on actual workplace exposures to ETS.[14/] Moreover, although many studies have monitored ETS constituent levels in certain areas of various workplaces, the ORNL and Hazelton studies actually measured

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[14/]Dr. Jenkins himself testified: "I might note that several of the speakers today have indicated the need to make assumptions concerning the relevant magnitude of home or workplace exposures, our study will provide hard data on that." (Transcript, Vol.VI, page 90, lines 9-13) [emphasis added]

ETS constituent levels in the breathing zones of individual employees. This latter measurement can only be obtained from personal air monitors worn by employees while they are at work. Philip Morris submits that the measurement of ETS constituent levels in the breathing zone of individual employees is the best method of accurately assessing actual workplace exposure. Most of the other studies, including the one by Mr. Repace cited at 59 FR 15991 (Table III-10), are based upon area monitoring, rather than personal monitoring of the employee's breathing zone. Moreover, Mr. Repace's study relies in significant part upon extrapolations, assumptions and modeling to try to predict the possible level of ETS constituents to which employees might be exposed in the workplace.

The "best available evidence" on ETS workplace exposure is the measurement of actual exposure obtained from personal monitoring in the breathing zone of employees at work. A "model," based upon assumptions, is on its face significantly less reliable. Moreover, studies based upon individual recollection of exposure, such as the two relied upon by OSHA to estimate risk in this rulemaking, are even less reliable than the available models. OSHA's estimate of the risk, if any, from exposure to existing levels of ETS in the workplace is thus not based upon the "best available evidence," as required by § 6(b)(5) of the OSHA Act.

b. Alleged health effects

Eleven of the 30 spousal smoking studies analyzed in the EPA Report included estimates of workplace ETS exposures in addition to exposure in the home environment. Fifteen separate risk estimates were presented in those studies. Thirteen of those 15 risk estimates were not statistically significant, thus not supporting the claim that an increased risk of lung cancer is associated with workplace ETS exposure. Moreover, if workplace data from these 11 studies were pooled in a meta-analysis, similar to the one conducted by the EPA on spousal smoking, the risk estimate would approximate 1.00, supporting no association between reported workplace exposures to ETS and lung cancer in non-smokers.[15/]

One of the most recent studies on the alleged association between workplace ETS exposure and lung cancer is the Brownson, et al. study, published in 1992.[16/] Although EPA was preparing its report on ETS, including a meta-analysis of the spousal smoking studies at that time, it ignored data from the Brownson study. Brownson and colleagues reported on results of a case-control study

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[15/]LeVois, M.E. and Layard, M.W., "Inconsistency Between Workplace and Spousal Studies of Environmental Tobacco Smoke and Lung Cancer," Regulatory Toxicology and Pharmacology 19: 309-316, 1994. Attached at Section V of this comment.

[16/]Brownson, R.C., Alavanja, M.C.R., Hock, E.T., and Loy, T.S., "Passive Smoking and Lung Cancer in Nonsmoking Women," American Journal of Public Health 82: 1525-1530, 1992. Attached at Section V of this comment.

of Missouri women who were lifetime nonsmokers or former smokers. The study is notable for its large sample size, since over 600 lung cancer cases were enrolled, more than 400 of whom were self-reported lifetime nonsmokers. The authors wrote: "In general, there was no elevated lung cancer risk associated with passive smoke exposure in the workplace." The authors of the study presented a risk estimate for the highest category of workplace exposure that did not reach statistical significance; they failed to present an overall workplace risk estimate.

Moreover, the extremely weak linkage between spousal smoking and chronic disease in nonsmokers has been acknowledged by Dr. Morton Lippmann, Chairman of the Science Advisory Board Committee that reviewed EPA's risk assessment on ETS. Speaking to reporters in April 1991, Dr. Lippmann stated that the risk attributed to ETS was "probably much less than you took to get here through Washington traffic" to attend the news conference held to discuss the Committee's recommendations on the first draft of the risk assessment.[17/]

More recently, on May 11, 1994, the authors of a recent Congressional Research Service (CRS) Report for Congress on the use of cigarette taxes to fund health care reform testified before the United States Senate Subcommittee on Clean Air and Nuclear

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[17/] See R.A. Taylor, "EPA Panel Reports Non-Smokers at Risk," The Washington Times, April 19, 1991, page A3 (Attached).

Regulation about the statistical basis for estimating the alleged health effects of passive smoking, particularly the EPA report.[18/] The authors concluded, "[O]ur evaluation was that the statistical evidence does not appear to support a conclusion that there are substantial health effects of passive smoking. This finding flows from an analysis of the statistical methodology employed in assessing such health effects." (page 13)

2. OSHA has failed to establish significant risk of material impairment from existing occupational exposure to ETS because, by OSHA's own account, the risk allegedly faced by exposed workers is indistinguishable from the purported risks faced by the general population

As noted above, OSHA's burden, as a threshold matter, is to show by substantial evidence that at existing levels of exposure in the workplace, a substance poses a significant risk of harm. In the case of ETS, however, OSHA has an additional task because the Agency contends "that exposure to ETS is common" (59 FR 15980) and that for the ETS constituent nicotine, "the range of average nicotine concentrations in office workplaces is very similar to that of homes." (59 FR 15994)

Where a substance is allegedly ubiquitous and exposure to that substance in the workplace is "indistinguishable" from exposure in the general population, as OSHA contends is the case

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[18/] A copy of the March 8, 1994 CRS Report, entitled "Cigarette Taxes to Fund Health Care Reform: An Economic Analysis," and the May 11, 1994 testimonial record are attached collectively.

with ETS, then the agency must determine the significance of regulating only workplace exposures to ETS before it can justify that such a regulation is "reasonably necessary or appropriate," as required by Benzene. The Agency must quantify the significance of the workplace exposure in terms of excess risk, if any, above and beyond that experienced by the general population, resulting from that exposure.

In OSHA's Hazard Communication Standard, 29 C.F.R. § 1910.1200, for example, OSHA properly chose not to regulate exposure to toxic substances in the workplace where exposure to these substances in the workplace was identical to exposures in non-occupational settings. Thus, the Hazard Communication provisions addressing "scope and application" provide, in pertinent part:

> (6) This section does not apply to:
>
> ...(vii) Any consumer product or hazardous substance, as those terms are defined in the Consumer Product Safety Act (15 U.S.C. 2051 et seq.) and Federal Hazardous Substances Act (15 U.S.C. 1261 et seq.) respectively, where the employer can demonstrate it is used in the workplace in the same manner as normal consumer use, and which use results in a duration and frequency of exposure which is not greater than exposures experienced by consumers;...

(1910.1200(b)(6)(vii)) Clearly, OSHA was acknowledging, in the context of the Hazard Communication Standard, that where exposures to "toxic" materials in the workplace are identical to exposures in

non-occupational settings, there is no need to regulate the workplace exposures.

The most recent example of an OSHA regulation which addressed potentially "ubiquitous" exposures that occur in both occupational and non-occupational settings arose in the case of the Bloodborne Pathogen Standard. There, OSHA analyzed significant risk in the workplace by stating:

> Clearly it is possible for workers with exposure to blood to become infected with the HBV [hepatitis B virus] by means other than occupational exposure. The virus can be transmitted sexually and by non-occupational exposure to blood. In addition, over fifty percent of the cases of HBV reported to the Centers for Disease Control in 1985 had no known risk factors (Ex. 6-217).
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> Several commentors viewed OSHA's estimates as being over-estimates of the true risk by stating that OSHA did not appropriately consider the fact that most healthcare workers who are infected with hepatitis B probably contracted their infection due to factors outside the workplace (Ex. 20-2879C). In fact, OSHA took measures to exclude the effect of high risk behaviors in healthcare workers by estimating the risk attributable to occupational exposure. <u>The risk attributable to occupational exposure is the difference between the risk faced by exposed workers and the background risk faced by the general population</u>. In order to remove that portion of HBV cases in healthcare workers that might be due to IV drug use or other known risk factors, the Agency subtracted from the healthcare worker risk the background (population) risk of HBV infection. [emphasis added]

(56 FR 64027)

If the foregoing test for differentiating "ubiquitous" risk is applied in this case, the result, according to OSHA's NPR,

would be zero risk attributable to occupational exposure to ETS. OSHA has asserted that "risk estimates based on residential exposures are expected to accurately reflect occupational risks in most workplaces and possibly underestimate the risk in some workplaces." (59 FR 15994) [emphasis added] However, as discussed elsewhere in this submission, recent studies report actual exposure levels in the workplace that are, in fact, much lower than exposure levels in the home. Nevertheless, even assuming for the sake of argument that home and workplace exposures are comparable, OSHA has failed to subtract non-occupational exposure from its risk estimate as was done in the Bloodborne Pathogens Standard. (29 CFR 1910.1030) In addition, OSHA made no attempt to quantify or substantiate this assumption for "some workplaces," so as to demonstrate the increased risk, if any, attributable to occupational exposure in those unique workplaces.

The Benzene Court also acknowledged the need to compare workplace risk with "every day" risk experienced by the general population in order to determine whether such workplace risk is "significant." That Court explained as follows:

> '[S]afe' is not the equivalent of 'risk-free.' There are many activities that we engage in every day--such as driving a car or even breathing city air--that entail some risk of accident or material health impairment; nevertheless, few people would consider these activities 'unsafe.' Similarly, a workplace can hardly be considered 'unsafe' unless it threatens the workers with a significant risk of harm. [emphasis added]

(448 U.S. at 642)

The Benzene Court recognized that certain activities which entail risks are nonetheless deemed insignificant because of the "every day" nature of these activities. Although driving a car, as cited by the Court, entails a lifetime accident risk of 20 in 1,000,[19/] the court deemed it insignificant due to the fact that it is an "every day" activity. OSHA has already admitted that, as with driving a car or breathing city air, "exposure to ETS is common" (59 FR 15980) and that for the biomarker nicotine, "the range of average nicotine concentrations in office workplaces is very similar to that of homes." (59 FR 15994) Therefore, exposure to ETS is an every day activity which allegedly entails a risk. One crucial difference, however, between the every day activities of driving a car and being exposed to ETS is that driving a car entails an accident risk of 20 in 1,000. With respect to ETS, however, OSHA has not shown and indeed cannot show that occupational exposure entails a risk of material health impairment.

Since the OSH Act limits OSHA to "reasonably necessary and appropriate" regulations, the Agency is constrained by its enabling legislation, as interpreted by the Courts and the Agency, to address those risks in the workplace that are significantly greater than the risks existing in the general population. Until existing workplace exposure to ETS is shown to be significantly greater than exposure in the general population, OSHA cannot

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[19/]Hallenbeck, W.H. and Cunningham, K.M., Quantitative Risk Assessment for Environmental and Occupational Health, Lewis Publishers (1986) page 3.

demonstrate that regulation of ETS in the workplace would be "reasonably necessary or appropriate."

B. The proposed standard's workplace smoking restrictions are not reasonably necessary to significantly reduce a significant risk

1. OSHA has not demonstrated that reducing the current OSHA permissible exposure limits for the constituents of ETS is necessary to reduce a significant risk

OSHA has failed to acknowledge that several of the constituents of ETS are already regulated by the Agency.[20/] OSHA currently regulates exposure to more than half of the constituents imputed to ETS, listed in OSHA's NPR (Tables III-6 and III-7). In previous rulemakings which addressed exposure to substances already covered by OSHA standards, the agency has consistently stated that prior to reducing the permissible exposure limit for this substance, it must first determine whether there is a significant health risk at the current exposure limit under the existing standards which would justify lowering this exposure limit further.

For example, in OSHA's preamble to its Cotton Dust Standard, the Agency determined that it should not lower the exposure limits for waste processing operations because there was

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[20/] Although portions of the Air Contaminants Standard were stayed by the U.S. Court of Appeals for the Eleventh Circuit in AFL-CIO v. OSHA, 965 F.2d 962 (11th Cir. 1992), OSHA continues to enforce the permissible exposure limits for air contaminants which were in place prior to 1992. (29 CFR 1910.1000 (Table Z-1))

not sufficient evidence that lowering the exposure limit beyond that already required by Table Z-1 would substantially reduce a significant risk. (50 FR 51120)[21/] OSHA, however, has made no such determination with respect to the constituents of ETS which are currently being regulated by the Agency.

Philip Morris submits that OSHA has already determined that occupational exposure to ETS is covered under existing standards. In an opinion letter from OSHA to Mr. H. Brandon on March 3, 1988 (attached), regarding occupational exposure to tobacco smoke, the Agency stated:

> The Occupational Safety and Health Administration (OSHA) does not have a standard on worker exposure to cigarette smoke in the workplace. OSHA does have an air contaminant standard, 29 CFR 1910.1000, Table Z-1, for the components of cigarette smoke, such as nicotine and carbon monoxide.

The letter concludes by recommending that Mr. Brandon call his area OSHA office for an inspection.

[21/] See also, 57 FR 24310 (OSHA's Final Rule on Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite), where OSHA explained its decision not to regulate occupational exposure to non-asbestiform ATA because: "employees exposed to talc containing ATA will be protected under the Air Contaminants Standard (29 CFR 1910.1001). OSHA believes that application of the talc limit in the Air Contaminants Standard . . . will protect exposed employees against a significant risk of nonmalignant disease." 57 FR 24310, 24327. Therefore, OSHA concluded that it could not regulate exposure to a substance where it could not demonstrate that reducing the exposure level specified in the Air Contaminants standard could protect employees from a significant risk of harm.

As stated above, before OSHA can attempt to reduce occupational exposure to ETS, the Agency must justify its proposed standard by showing that there is a significant risk of material impairment to health from occupational exposure to ETS at the current Air Contaminants exposure levels and that this risk can be substantially reduced by lower exposure levels.

2. OSHA may not regulate insignificant risks

Under Benzene and its progeny, OSHA is precluded from regulating insignificant risks and only has authority to regulate significant risks to the point they become insignificant. To set a zero exposure limit, according to Benzene, OSHA must first demonstrate that any level of the substance, no matter how minute the exposure, poses a significant risk. (448 U.S. at 645) Only by actually quantifying and assessing the potential risk posed by various levels of ETS could OSHA even attempt to make such a demonstration.

In Benzene, OSHA set a permissible exposure limit for benzene at the lowest feasible level and banned all dermal contact with the substance without first demonstrating that any level, no matter how minute the exposure, posed a significant risk. OSHA relied on its carcinogen policy in effect at that time, which provided that "in the absence of definitive proof of a safe level, it must be assumed that any level above zero presents some

increased risk of cancer." (448 U.S. at 635-636) [emphasis added] OSHA contended that under that policy, if a safe level could not be proven, it was justified in regulating benzene to the lowest feasible level, which, in some cases, would be an outright ban.

Rejecting this aspect of OSHA's carcinogen policy, the Supreme Court made it very clear that OSHA's authority to promulgate safety and health standards is limited. The Court pointed out that "both the language and the structure of the [OSH] Act, as well as the legislative history, indicate that it was intended to require the elimination, as far as feasible, of significant risks of harm." (Id. at 641)

According to the Court, the legislative history supports the conclusion that "Congress was concerned, not with absolute safety, but with the elimination of significant harm." (448 U.S. at 646) The Court commented that "Congress specifically amended [29 U.S.C. § 655(b)(5)] to make it perfectly clear that it does not require [OSHA] to promulgate health standards that would assure an absolutely risk-free workplace." (48 U.S. at 646-647) The Court explained further:

> By empowering the Secretary to promulgate standards that are 'reasonably necessary or appropriate to provide safe or healthful employment and places of employment,' the Act implies that, before promulgating any standard, the Secretary must make a finding that the workplaces in question are not safe. But 'safe' is not the equivalent of 'risk-

> free.' There are many activities that we engage in every day -- such as driving a car or even breathing city air -- that entail some risk of accident or material health impairment; nevertheless, few people would consider these activities 'unsafe.' Similarly, a workplace can hardly be considered 'unsafe' unless it threatens the workers with a significant risk of harm.
>
> Therefore, before he can promulgate any permanent health or safety standard, the Secretary is required to make a threshold finding that a place of employment is unsafe -- in the sense that significant risks are present and can be eliminated or lessened by a change in practices.

(448 U.S. at 642) [emphasis added]

The Court reasoned that to allow OSHA to regulate a substance, no matter how minute the exposure, would constitute an unconstitutional delegation of legislative power:

> In the absence of a clear mandate in the Act, it is unreasonable to assume that Congress intended to give the Secretary the unprecedented power over American industry that would result from the Government's view of [29 U.S.C. §§652(8) and 655(b)(5)], coupled with OSHA's cancer policy. Expert testimony that a substance is probably a human carcinogen -- either because it has caused cancer in animals or because individuals have contracted cancer following extremely high exposures -- would justify the conclusion that the substance poses some risk of serious harm no matter how minute the exposure and no matter how many experts testified that they regarded the risk as insignificant. That conclusion would in turn justify pervasive regulation limited only by the constraint of feasibility. In light of the fact that there are literally thousands of substances used in the workplace that have been identified as carcinogens or suspect carcinogens, the Government's theory would give OSHA power to

impose enormous costs that might produce little, if any, discernible benefit.

(448 U.S. at 645) [emphasis added][22/] The Court thus concluded that an interpretation of the OSH Act that would give OSHA such "unprecedented power" to ban "any level" of a carcinogen, "no matter how minute the exposure," with "little, if any, discernible benefit" was both unreasonable and unlawful, absent a clear legislative mandate.[23/]

The Court also noted that § 6(b)(5) of the OSH Act suggests that a ban is unwarranted unless it can be established that a significant risk exists at any level of exposure. "While [§

[22/]In his concurring opinion, Chief Justice Burger made this same point, stating, "Perfect safety is a chimera; regulation must not strangle human activity in the search for the impossible." (448 U.S. at 664)

[23/]The Court also pointed out that OSHA's goal of eliminating all carcinogens from the workplace was an unconstitutional usurpation of legislative power. Specifically, the Court concluded:

> If the Government were correct in arguing that neither §3(8) nor §6(b)(5) requires that the risk from a toxic substance be quantified sufficiently to enable the Secretary to characterize it as significant in an understandable way, the statute would make such a 'sweeping delegation of legislative power' that it might be unconstitutional . . . . A construction of the statute that avoids this kind of open-ended grant should certainly be favored.

(448 U.S. at 646 (quoting A.L.A. Schechter Poultry Corp. v. United States, 295 U.S. 495, 539, 79 L.Ed. 1570, 55 S.Ct. 837 (1935) and Panama Refining Co. v. Ryan, 293 U.S. 388, 79 L.Ed. 446, 55 S.Ct. 241 (1935)))

6(b)(5)] requir[es] the Secretary to promulgate the standard that 'most adequately assures [to the extent feasible] . . . that no employee will suffer material impairment of health or functional capacity,' [it] also contains phrases implying that the Secretary should consider differences in degrees of significance rather than simply a total elimination of all risks." (Id. at 643, n. 48) According to the Court, a workplace ban on a substance like ETS requires more than a finding by OSHA that a significant risk exists at current, unregulated levels. The Agency must determine that the risks at any levels, even minute levels, are significant.

In defense of its standard in the Benzene case, OSHA argued that the language of § 3(8) merely means that the Agency is not required to eliminate insignificant risks, but that § 6(b)(5) called for the regulation of any risks posed by toxic or harmful physical substances. The Court responded that such "interpretation [was] at odds with Congress' express recognition of the futility of trying to make all workplaces totally risk free." (448 U.S. at 650) Summing up OSHA's limited rulemaking authority and referring to the Agency's recognition of not being required to regulate insignificant risks, the Court asserted: "It is entirely consistent with this interpretation to hold that the Act also requires the Agency to limit its endeavors in the standard-setting area to eliminating significant risks of harm." (448 U.S. at 651) As a result, OSHA's standard-setting authority is expressly limited to eliminating or reducing significant risks. It is not within the

realm of OSHA's authority to regulate exposure to substances which it has failed to establish on the basis of substantial evidence pose a significant risk of material harm. Thus, OSHA may not regulate insignificant risks.

Further addressing the OSH Act's legislative history, the Court noted that "Congress repeatedly expressed its concern about allowing [OSHA] to have too much power over American industry." (448 U.S. at 651) For instance, Congress refused to give OSHA the power to unilaterally close plants because of an alleged imminent danger, and narrowly circumscribed OSHA's power to issue emergency temporary standards. (Id.) According to the Court:

> This effort by Congress to limit the Secretary's power is not consistent with a view that the mere possibility that some employee somewhere in the country may confront some risk of cancer is a sufficient basis for the exercise of the Secretary's power to require the expenditure [or cost to industry] of hundreds of millions of dollars to minimize that risk.

(448 U.S. at 651-652) [emphasis added] In sum, the Court's review of the legislative history of the federal Act clearly demonstrates that OSHA's authority to regulate toxic materials or harmful physical agents is limited to significant risks.

Directly in response to the Supreme Court's decision in Benzene, OSHA amended its cancer policy to no longer require exposure reduction to the "lowest feasible level." (46 FR 5881)

The new policy recognizes OSHA's duty to establish a significant risk and its limited authority to reduce or eliminate that risk.

In addition, subsequent to the decision in Benzene, a majority of the Supreme Court recognized the two pronged significant risk test. (See, American Textile Manufacturers Institute, Inc. v. Donovan, 452 U.S. 490, 505, n. 25, 69 L. Ed.2d 185, 101 S. Ct. 2478 (1981)) The Courts of Appeals have elaborated on these requirements and how they prevent OSHA from regulating insignificant risks.

In International Union, UAW v. Pendergrass, 878 F.2d 389 (D.C. Cir. 1989), the court held that OSHA failed to sufficiently explain its finding of insignificant risk of an exposure level of 1 ppm for formaldehyde. In promulgating the standard, the Agency set a permissible exposure limit (PEL) of 1 ppm in conjunction with a short-term exposure limit (STEL) of 2 ppm. It reasoned that the PEL combined with a STEL was likely to decrease risks from formaldehyde exposure to a level at which those risks are insignificant. One issue in the case centered on why OSHA chose the 1 ppm exposure level as posing an insignificant risk. As it turned out, the Agency was confronted with conflicting estimates of risk at particular levels. Rather than automatically setting a PEL according to the higher risk estimate (thereby requiring a lower PEL), OSHA accorded more weight to the lower risk estimate and implemented a PEL and a STEL so as not to regulate insignificant

risks. While the Court remanded the case to OSHA for reconsideration of its risk calculations, the case illustrates OSHA's recognition that it cannot regulate insignificant risks.

The D.C. Circuit faced a similar issue in Building & Construction Trades Department, AFL-CIO v. Brock, 838 F.2d 1258 (D.C. Cir. 1988), by examining whether OSHA had successfully carried its burden of proving that a total ban was reasonably necessary to reduce a significant risk. In that case, OSHA banned spraying of any and all products containing asbestos. OSHA also claimed to find support for its ban in the fact that both the EPA and the State of California had imposed similar bans. However, the Brock court found that evidence in the rulemaking record indicated that the modern process of encapsulating spray-on asbestos products ensures that asbestos fibers are not released on application. According to the court, OSHA failed to refute that claim.

In striking down the ban on spraying of any and all products containing asbestos, the Court found the ban to be overly broad, stating that OSHA failed to meet the substantial evidence standard imposed by the OSH Act. Although the court did not specify that OSHA failed to demonstrate the requirement that the ban be reasonably necessary, it is clear that the Agency could not show that completely prohibiting the spraying of any and all forms of asbestos was reasonably necessary to reduce a significant risk of material health impairment.

In United Steelworkers of America, et al. v. Marshall, 647 F.2d 1189 (D.C. Cir. 1980), a decision issued immediately after Benzene, the court reviewed the significant risk requirements. The Steelworkers court cited Benzene for the proposition "that Congress had not mandated OSHA to seek an absolutely risk-free workplace or to require industry to eliminate even insignificant risks of harm so long as the effort is not technologically impossible or financially ruinous." (Id. at 1246, citing Benzene, 448 U.S. at 641) [emphasis added] Furthermore, the Steelworkers court asserted that § 3(8) appears to require OSHA to establish a significant risk at the PEL that it sets.

> In mentioning this second aspect of the threshold requirement -- whether the significant harm at the current level can be eliminated or lessened by a change in the PEL -- the plurality implies that § 3(8) requires OSHA to prove by specific evidence the level of risk at the new PEL, as well as the current PEL. The plurality leaves this point somewhat unclear, but in any event, a requirement of such proof would seem to follow from the second statutory provision governing OSHA's toxic agents standards, § 6(b)(5).

(Id. at n. 85)

The court touched upon the exact issue which becomes relevant in attempting to ban a substance. OSHA may only regulate significant risks. It is neither required nor authorized to regulate insignificant risks. It may not ban a substance without determining that any level of exposure, no matter how minute, poses a significant risk.

The Second Circuit has also addressed OSHA's lack of authority to regulate insignificant risks. In Pratt & Whitney Aircraft, et al. v. Secretary of Labor, 715 F.2d 57 (2d Cir. 1983), an enforcement proceeding, the court asserted that the "test employed by the [Review] Commission 'would permit this safety standard to be applied to conditions posing insignificant risks that are beyond the scope of the [OSH] Act.'" Id. at 59, quoting Pratt & Whitney Aircraft, et al. v. Secretary of Labor, 649 F.2d 96, 104 (2d Cir. 1981). The Court supported this assertion by referring to Benzene. According to the Second Circuit, and others, OSHA is prohibited not only from promulgating a new standard that reduces only insignificant risks, it may not enforce heretofore unchallenged or existing standards that reduce insignificant risks.

In addition to the courts, OSHA itself has acknowledged that the OSH Act limits its authority to requiring elimination of significant risks only. (Glycol Ethers Proposed Rule, 58 FR 15526, 15547, citing, Benzene, 448 U.S. at 644, n. 49) (See also, Cadmium Final Rule, 57 FR 42102, 42103; Air Contaminants for Maritime, Construction, and Agriculture Industry Sectors, Proposed Rule 57 FR 26002, 26034; Bloodborne Pathogens Final Rule, 56 FR 64004, 64005; Benzene Final Rule, 52 FR 34460, 34465)

More recently, in the Final Rule for Personal Protective Equipment for General Industry, published April 6, 1994, 59 FR 16334, OSHA recognized the limits the OSH Act places on its

rulemaking authority. In the preamble, the Agency asserts that it "has long followed the teaching that § 3(8) of the OSH Act requires that, before it promulgates 'any permanent health or safety standard, [it must] make a threshold finding that a place of employment is unsafe -- in the sense that significant risks are present and can be eliminated or lessened by a change in practices.'" (59 FR at 16357, citing Benzene, 448 U.S. at 642) Moreover, the Agency, citing § 6(a) of the Act, asserted that Congress' instruction to OSHA to summarily adopt national consensus and existing federal standards establishes a reference point "concerning the least an OSHA standard should achieve." (59 FR at 16357) Reasoning from this point, OSHA determined that it "is precluded from regulating insignificant safety risks or from issuing safety standards that do not at least lessen risks in a significant way." (Id.) [emphasis added]

On June 2, 1994, OSHA, in its publication of a Proposed Rule for Longshoring and Marine Terminals, expressed the same reasoning and came to the same conclusion. (59 FR 28594) OSHA is precluded from regulating insignificant risks. (58 FR 28598) As OSHA explained, based on both judicial and Agency interpretation, "the OSH Act sets clear and reasonable limits for Agency rulemaking action." (Id.; 59 FR at 16357)

OSHA has also recognized its limited authority in the promulgation of health standards. In fact, the Agency acknowledged

this limitation to also preclude it from regulating a substance below levels at which the risks from exposure become insignificant. The promulgated standards for air contaminants in maritime, construction, and agriculture industry sectors, 57 FR 26002; bloodborne pathogens, 56 FR 64004; cadmium, 57 FR 42102; and benzene, 52 FR 34460, all expressly recognize OSHA's lack of authority to regulate insignificant risks.

In addition, the Agency, in its Air Contaminants Final Rule, asserted that in promulgating a health standard its aim "is to set the lowest feasible level necessary to eliminate significant risks." (54 FR at 2332, 2361; see also, Proposed Rule for Maritime, Construction, and Agriculture Sectors, 57 FR at 26034) The Agency draws this "aim" from the significant risk requirement of Benzene and the court's language in Cotton Dust that "a cost benefit analysis is not required by statute because a feasibility analysis is." (452 U.S. at 531, n. 32) Thus, the Agency recognizes that its goal is not to achieve the lowest feasible level of exposure. Rather, the OSH Act places dual limitations on OSHA's authority to promulgate standards. First, the standard must be technologically and economically feasible. Second, even if a lower standard is feasible, it must be necessary to eliminate or reduce a significant risk. In other words, OSHA may regulate a risk to the point it is no longer significant but no further. If a level of exposure to a substance like ETS exists that does not

pose a significant risk of material health impairment, OSHA cannot promulgate a ban or otherwise regulate that insignificant risk.

OSHA has consistently acknowledged its limited authority through the painstakingly detailed analysis it undertakes in establishing significant risks and setting PELs. In the preamble to the final Benzene Standard, OSHA concluded that after "reviewing all the evidence and comments . . . the final standard is carrying out Congressional intent within the limits of feasibility and does not attempt to reduce insignificant risks." (52 FR at 34463) [emphasis added] See also, proposed rule for amendment to Air Contaminants Standard for Maritime, Construction, and Agricultural Industry Sectors, 57 FR 26002, 26049 ("OSHA is also confident that it is not attempting in this rulemaking to reduce exposure to insignificant levels") (emphasis added). The Agency made this statement after describing the threshold requirement of the Benzene decision, i.e., to establish that existing levels of benzene exposure posed a significant risk.

Similarly, in the preamble to the Cadmium Standard, OSHA asserted that by setting "a PEL of 5 ug/m^3, [it was] assured that the Agency is not regulating an insignificant excess risk of cancer or kidney damage." The statement again indicates OSHA's recognition of two primary limitations on its authority to regulate a substance stemming from Benzene and its progeny. First, OSHA

must not regulate insignificant risks. Second, it is only the significant excess risk that the Agency is authorized to regulate.

Finally, in the preamble for the Bloodborne Pathogens Final Rule, OSHA also acknowledged its limited authority under the OSH Act. "OSHA believes the standard for bloodborne pathogens will reduce risks of HBV infection and material impairment of health from 83 to 113 per 1,000 to 3 to 5 per 1,000, the Agency is carrying out the Congressional intent and is not attempting to reduce insignificant risks." (56 FR at 64037) [emphasis added] Through this statement, OSHA estimates the excess risks remaining after implementation of the Bloodborne Pathogens Standard. In light of the Agency's policy of designating a risk of 1 in 1000 or higher as significant, it appears that a significant risk remains. Nevertheless, OSHA went to great lengths through this quantification to demonstrate that it was not regulating an insignificant risk.

In sum, the Supreme Court, every court of appeals that has addressed the issue, and OSHA have consistently interpreted §§ 3(8) and 6(b)(5) of the OSH Act as placing limits on the rulemaking authority of OSHA to regulate only significant risk.

3. OSHA has acknowledged that evidence is lacking to establish that any level of ETS in enclosed work areas poses a significant risk

In the NPR, OSHA expresses its position that the proposed standard "reduces significant risk to only a small percentage of the current risk. To the extent that there are failures of enforcement of the smoking limitation and of the ventilation system, the risk will not be totally eliminated." [emphasis added] (59 FR 16001) OSHA then proclaims, "Since there is no definition of, nor an established method for quantifying exposure, it is not possible to determine a 'dose limit' that would eliminate significant risk. Even if that were possible, it is not clear it would be the correct policy approach." (59 FR 16001)

OSHA's position absolutely defies the Supreme Court's ruling in Benzene that the Agency is only authorized to reduce a significant risk. According to the NPR, OSHA believes that it may "totally eliminate" risk, whether or not that risk is significant, where it is not possible to determine the "dose limit" that would eliminate significant risk. OSHA fails to cite to any authority for this belief and Philip Morris submits that no such authority exists. Nothing in Benzene, or any of the court cases interpreting Benzene, frees OSHA from the significant risk requirement when a so-called "dose limit" cannot be determined.

OSHA's inability to designate the point at which a risk becomes insignificant does not permit it to ban exposure to a particular substance. Rather, OSHA's authority is limited to regulating risks that it can establish on the basis of substantial evidence are significant. Nevertheless, despite OSHA's acknowledged lack of evidence on what amount of ETS causes the alleged significant risk, the Agency attempts to set a zero exposure limit for nonsmoking employees.

OSHA did not even attempt to establish that any level of ETS, no matter how minute the exposure, poses a significant risk. In fact, there is scientific debate as to whether there may be a level of ETS at which any alleged risk would be insignificant. Dr. Steven Bayard of the EPA, who was a coauthor of the Agency's ETS risk assessment, which OSHA relies upon so heavily, testified before the Maryland Occupational Safety and Health (MOSH) Advisory Board on December 16, 1993, that he thought that a threshold level did exist below which any risk posed by ETS is insignificant. When asked by a Board Member: "[D]o you believe that there is a level of environmental tobacco smoke at which a person who's exposed will not suffer material impairment of health or functional capacity?" Dr. Bayard responded, "The answer is, certainly." (Transcript, Vol. II, page 297, lines 2-7) [emphasis added] Dr. Bayard responded further:

> And some of these people that are coming down with these diseases are just highly sensitive populations, and so you're not dealing with a normal population, I don't think. I think

you're dealing with normal people and then you're dealing with highly sensitive people.

So, yes, certainly.

Id. at lines 13-18.

Mr. James Repace of EPA also believes that a threshold of exposure exists below which there is no significant risk of material health impairment. Mr. Repace contributed to the risk assessment in the EPA Report and is heavily relied upon by OSHA in its NPR. He testified before the MOSH Advisory Board on December 9, 1993 that 7 or 8 nanograms of nicotine per cubic meter is "an insignificant or trivial risk." Transcript, Vol. I, page 180, lines 5-13.

In addition, the March 1994 Congressional Research Service (CRS) Report for Congress on the use of cigarette taxes to fund health care reform found plausible the existence of an exposure threshold. The authors stated:

> The existence of an exposure threshold for disease below which many passive smokers fall is not implausible. Some organisms have the capacity to cleanse themselves of some level of contaminants. It is for this reason that public policy usually does not insist that every unit of air or water pollution be removed from the environment; the damage of low levels of pollutants is sufficiently small (through the self-cleansing process) that removal is not cost effective. In fact, strongly nonlinear relationships in which health effects rise with the square of exposure, and more, have been found with respect to active smoking (See, Surgeon General's Report, 1989, p. 44). Were these

> relationships projected backward to construct the lower (unknown) portion of the health effects/physical damage function, the observed relationship might lead researchers a priori to expect no empirical relationship. Thus, the issue raised by this potential break in the causative chain is whether researchers should expect to find a significant relationship between passive smoking and health effects. (p.45) [emphasis added]

Despite this acknowledged lack of data to support the assumption that any level of ETS poses a significant risk, and despite the data that a threshold level may exist below which the risk, if any, posed by ETS is insignificant, OSHA proposes to mandate a zero exposure limit for non-smoking employees and thereby allegedly guarantee absolute safety. According to Benzene, OSHA may not rely on such an unsupported assumption or policy.

4. OSHA's proposed zero exposure limit does not significantly reduce the alleged significant risk because of non-workplace "background" exposure

In addition to holding that OSHA may not regulate insignificant risk, the Benzene Court asserted that OSHA standards must significantly reduce the hazard risk.

Exposure to a substance in locations other than an employee's workplace, such as in the home or other "background" exposure sites, may prevent a standard from achieving such a significant reduction. OSHA itself implicitly recognized this when

regulating benzene. Instead of banning the substance outright, OSHA "began with a 1 ppm level, selected in part to ensure that employers would not be required to eliminate benzene concentrations that were little greater than so-called background exposures experienced by the population at large." (448 U.S. at 650) [emphasis added] The National Institute for Occupational Safety and Health (NIOSH) had recommended the 1 ppm permissible exposure level ("PEL") to OSHA, "because any lower standard might require the elimination of the small amounts of benzene (in some places up to 0.5 ppm) that are normally present in the atmosphere." (448 U.S. at 621 n.14) [emphasis added] Thus, through other regulatory efforts, both OSHA and NIOSH have recognized that lowering of PELs must stop when no further significant reduction of risk would be achieved because of "background" exposures.

The D.C. Circuit, in Public Citizen Health Research Group v. Tyson, 796 F.2d 1479 (D.C. Cir. 1986) ("Ethylene Oxide"), acknowledged that "background" exposure is a factor that OSHA must consider in determining whether a standard significantly reduces the risk. The Court observed that for a short term exposure limit (STEL) to achieve a risk reduction, workplace ethylene oxide exposure patterns had to be characterized by "low background levels and high intermittent exposures": i.e., workplace exposure levels had to be quantitatively greater and qualitatively different from those experienced by the general public in non-workplace settings. (796 F.2d at 1506) Since OSHA had "made no findings at all on

patterns of exposure," the Court held that OSHA "improperly assumed that exposure scenarios will eliminate the need for a STEL." (796 F.2d at 1507) [emphasis added] The Court remanded the standard to OSHA for further consideration, stating: "On remand, we expect the Agency to ventilate the issues on this point thoroughly and either adopt a STEL or explain why empirical or expert evidence on exposure patterns makes a STEL irrelevant to controlling long-term average exposures." (Id.) [emphasis added]

Similarly, before OSHA may ban smoking from the work areas of enclosed workplaces, it must address patterns of "background" exposure: i.e., the impact, if any, on employee health of ETS exposure outside the workplace. This means finding "substantial evidence" to support the proposition that any ETS increase, no matter how minute, in workplace exposure -- above and beyond the employee's non-work "background" exposure levels -- will significantly increase the risk of material health impairment to a significant level. Only by showing this can OSHA demonstrate that a zero exposure workplace limit is reasonably necessary to significantly reduce that significant risk. OSHA has not even attempted to address this issue.

5. OSHA failed to demonstrate that its choice from among alternative regulations was reasonably necessary to significantly reduce the alleged risk

a. OSHA must adequately consider alternative regulations and offer a reasonable explanation for its choice from among those alternatives

The federal courts have explained that in promulgating an occupational safety and health standard, OSHA must consider degrees of risk and alternative kinds or levels of regulation. The Supreme Court in Benzene noted that it is implicit in the OSH Act that "the Secretary should consider differences in degrees of significance rather than simply a total elimination of all risks." (Id. at 643 n.48) [emphasis added]

Similarly, in Dry Color Manufacturers' Ass'n v. Department of Labor, 486 F.2d 98 (3rd Cir. 1973), the Third Circuit Court of Appeals ruled that OSHA must provide "at least a general explanation as to why the procedures prescribed were chosen in light of . . . the alternative kinds of regulations considered by OSHA." Furthermore, OSHA must consider alternative approaches that could achieve the same goals, in this case the reduction of significant risk, with minimal economic impact. Pursuant to Executive Order 12291, OSHA must conduct a Regulatory Impact Analysis (hereinafter "RIA") which includes:

> a description of alternative approaches that could substantially achieve the same regulatory goal at lower costs, together with an analysis of the potential benefit and costs, and a brief explanation of the legal

> reasons why such alternatives, if proposed, could not be adopted;

(Executive Order 12291, § 3(d)(4)) Moreover, OSHA is under a statutory obligation to perform a Regulatory Flexibility Analysis (hereinafter "RFA") "describ[ing] the impact of the proposed rule on small entities." (5 U.S.C. 603(a)) The RFA must include:

> [A] description by (sic) any significant alternatives to the proposed rule which accomplish the stated objectives of applicable statutes and which minimize any significant economic impact of the proposed rule on small entities.

(Id. at 603(c))

In the instant situation, OSHA has failed to satisfy its obligation to examine alternative kinds of regulation under this proposed standard and to explain the choice it made from among the available alternatives.

b. OSHA has failed to offer a reasonable explanation for its choice from among sufficiently effective alternative regulations

As stated previously, OSHA is charged with eliminating exposures that purportedly create significant risk, not any possible risk. Philip Morris submits that OSHA has failed to consider alternatives, including the physical separation of smokers and nonsmokers, providing adequate ventilation, or even restricting smoking to designated areas with negative pressure ventilation (but

without direct exhaust to the outdoors), which would reduce exposure and hence any alleged significant risk to a level at which it is no longer significant. Choosing to ignore these alternatives, OSHA failed to offer a reasonable explanation for choosing the most restrictive alternative, i.e., a ban on smoking in areas where employees work.

c. Alternative regulations would be less burdensome than the proposed standard

In the NPR, OSHA states "that control of pollutants at the source is the most effective strategy for maintaining clean indoor air . . . . In the case of ETS, this means restricting smoking to separately ventilated spaces." (59 FR 15968)

Philip Morris submits that the proposed standard places a significantly greater burden on employers, with no corresponding reduction in risk, than would be imposed by equivalent effective alternatives which OSHA failed to consider. The proposed standard requires that designated smoking areas be "enclosed and exhausted directly to the outside." This requirement may be cost prohibitive for many employers, depending on the facility and size of the company. In addition, the requirement that smoking room air be "maintained under negative pressure sufficient to contain tobacco smoke within the designated area" would likely burden most employers with the expense of purchasing and installing the duct work necessary to direct smoking room air to the outside, the

expense of purchasing and installing fans capable of maintaining the required negative pressure, the expense of energy to operate the fans, and the expense to heat or cool intake air to replace the air exhausted from the smoking room. By comparison, what are the costs of monitoring to ensure compliance with a permissible exposure limit, policing employee habits to ensure separation of smokers and nonsmokers, or maintaining the general ventilation rate at applicable standards?

OSHA failed to adequately estimate the costs of alternatives to the proposed standard, something which it is required to do.[24/] Philip Morris submits that none of the alternative controls that OSHA failed to consider could be any more burdensome than the proposed standard. Although OSHA may believe that a ban is the easiest and least costly method of reducing ETS exposure, this belief is premised on an assumption that the employer does not want to permit any form of workplace smoking. For the employer who desires to accommodate the preferences of both its smoking and nonsmoking employees, the proposed regulatory scheme for ETS is unduly burdensome and costly. Other options, such as those discussed below, must be considered.

[24/]For example, in OSHA's advanced notice of proposed rulemaking on the reevaluation of the Cotton Dust Standard, OSHA asked for comments on the question of whether there are more "cost effective ways of reducing cotton dust related illnesses." (47 FR 5906, 5909 (1982))

(1) General workplace smoking accommodation

For many years, large and small groups of smokers and nonsmokers have worked together. If disputes arose, settlements were reached through discussion and negotiation. Many successful companies have adopted an informal approach to workplace smoking using a minimal level of administrative structure and supervision to address individual complaints. Complaints about exposure to tobacco smoke typically have been addressed by employers through effective, common sense responses such as:

1. Increased outdoor air ventilation to levels specified in local building codes, or more recently, to levels such as those specified in ASHRAE Standard 62-1989 "Ventilation for Acceptable Indoor Air Quality;"[25/]

2. Relocation of a workstation;

3. Grouping of smokers and nonsmokers;

4. Partitions in "open" office settings;

5. Use of fans or vents as may be appropriate;

6. Implementation of a smoking policy either designed to accommodate smokers and nonsmokers or reached through the collective bargaining process.

[25/] ASHRAE Standard 62-1989 "Ventilation for Acceptable Indoor Air Quality" specifies "minimum ventilation rates and indoor air quality that will be acceptable to human occupants and are intended to avoid adverse health effects." Submitted in response to OSHA's RFI. (Ex. 3-1074)

In fact, the most recent Society for Human Resource Management survey of human resources executives indicates that 85% of companies adopted workplace smoking policies.[26/] The percentage of companies implementing smoking policies has risen dramatically compared to the 36% of companies with policies in 1986. The large increase is evidence that smoking in the workplace is an issue designated for resolution through the employer/employee relationship.

Dr. Antonia C. Novello, Special Representative of Health and Nutrition, National Institutes of Health, testified before Congress regarding H.R. Bill 3434 on February 7, 1994, and elaborated on workplace smoking policies. (Attached) She acknowledged that 85% of private companies adopted policies to restrict or ban smoking. Moreover, she noted that 59% of worksites with 50 or more employees implemented formal policies that prohibited or severely restricted smoking. Clearly, these figures indicate that employers have addressed the issue of workplace smoking. Through the employment relations process, companies have reached mutually agreed upon policies tailored to specific worksites. The policies address issues unique to each environment and workforce and ensure satisfactory workplace conditions.

[26/] "SHRM-BNA Survey No. 55: Smoking in the Workplace: 1991," Bulletin to Management, August 29, 1991, pages 1-16.

Philip Morris submits that such measures have been effective in addressing complaints about ETS and generally require few additional costs or expenditures from the employer. These remedies are based on the concept of accommodation for both smokers and nonsmokers and the belief that cooperation among smokers and nonsmokers can be fostered in the workplace. By failing to address these and other alternatives to the proposed standard, OSHA ignores the quickest, most cost effective and beneficial methods of addressing indoor air constituents.

(2) Increased general ventilation

Adequate ventilation, such as that specified in ASHRAE Standard 62-1989 ("Ventilation for Acceptable Indoor Air Quality"), can effectively and efficiently reduce levels of ETS constituents as well as levels of numerous other substances that may contribute to the quality of indoor air.

In the case of indoor office work environments, the ASHRAE Standard stipulates a minimum outside air ventilation rate of 20 cfm/person, which allows for a moderate amount of smoking. This ventilation rate, which has been adopted by various building code organizations and many municipalities through the U.S., is designed to address all kinds of substances in indoor air, including tobacco smoke. Therefore, with a 20 cfm/person ventilation rate, moderate smoking activity is accommodated.

Implementation of a generic ventilation-based indoor air quality standard, such as that recommended by ASHRAE, offers a comprehensive solution to poor indoor air quality. Adequate supply (outdoor) air intake and its appropriate distribution throughout occupied spaces serves to dilute and/or remove a wide range of substances potentially in the indoor air, including volatile organic compounds, carbon monoxide, carbon dioxide, constituents of ETS, radon and biologicals. Even though workplace office configurations are infinitely varied, the ventilation solution remains constant as the work activities of building tenants change over time.

(3) Simple separation of smokers and nonsmokers

No data exist which suggest that the dedicated smoking lounge, as proposed in the NPR, will significantly reduce exposure to ETS-related constituents beyond reductions achieved by appropriate ventilation and the simple separation of smokers from nonsmokers.

Recent studies on ETS constituent levels aboard commercial aircraft, including a 1989 study performed for the U.S. Department of Transportation, indicate the effectiveness of simple separation of smokers and nonsmokers in the minimization of ETS exposures. (See Ex. 3-1074) Similarly, Proctor (1987) monitored ETS constituents before and after a smoking ban on public transportation in the United Kingdom. While nicotine

concentrations decreased from 7 ug/m^3 (micrograms per cubic meter) to 3 ug/m^3 in nonsmoking compartments after the ban, particulate and CO levels remained unchanged. (See Ex. 3-1074) This suggests that ETS contributions to levels of particulates and CO are not significant. Thus, the submission to the OSHA RFI public docket from R.J. Reynolds observes:

> Any assessment of the need for measures to supplement a generic standard with special provisions such as mandated smoking lounges with separate exhaust or a smoking ban to reduce further any residual levels of ETS would plainly reveal that such measures would only result in an insubstantial reduction of an already de minimis exposure level. Accordingly, the imposition of such measures as mandated smoking lounges or smoking bans would be impermissible under the Supreme Court's direction that OSHA is authorized only to eliminate significant risks and may not seek the 'regulation of insignificant risks.' (Ex. 3-1086)

The aforementioned data illustrate the extremely low levels of ETS constituents that are supposedly "transferred" from smoking to nonsmoking areas, even under conditions involving a shared ventilation system. As discussed in other sections of this comment, reported data indicate that ETS constituents in nonsmoking areas in buildings where smoking is permitted are often only slightly above the limits of detection, and often indistinguishable from levels that can be found in buildings in which smoking is altogether prohibited. Philip Morris submits that simple

separation of nonsmokers and smokers can adequately minimize nonsmoker exposure to environmental tobacco smoke.

(4) Designated smoking areas operating at negative pressure

Another alternative method of regulating workplace smoking without imposing excessive costs on employers would be to eliminate the requirement from the proposed standard that designated smoking areas have a separate exhaust system to the outside of the building. If designated smoking areas were simply required to operate at negative pressure with respect to the rest of the indoor space, employers would not have the additional cost of creating this separate exhaust system. Philip Morris submits that the available data indicate that in such situations the nonsmoker exposure to ETS will be, at most, minimal. See discussion elsewhere in this comment. The designation of smoking areas with negative pressure could be accomplished at less cost than OSHA's proposed standard.

From an engineering and policy perspective, designated areas (including, but not limited to enclosed smoking rooms) operated under negative pressure have been embraced in a variety of contexts. For example, many restaurants operate designated areas such as the kitchen and smoking areas under negative pressure. Hospitals frequently operate certain rooms or wards, such as a TB ward, under negative pressure. Manufacturing firms utilize

negative pressure to assist in developing and maintaining "clean rooms" and other controlled environments. Hayward, et al., in their article "Effectiveness of Ventilation and Other Controls in Reducing Exposure to ETS in Office Buildings" (November 1993), note that the pressure relationships governing air movement in the building relative to smoking areas are a component of a mechanism to control recirculation of, or exposure to, ETS. (Submitted at Section XI of this comment.)

With respect to OSHA's concerns regarding the recirculation of air from smoking areas to nonsmoking areas, Philip Morris submits that the published literature also demonstrates that recirculation is a viable option over the separate exhaust requirement imposed by OSHA.

For example, in 1991, Hedge, et al., reported results of ETS constituent measurements taken in buildings with different smoking policies. For most constituents, the researchers reported no significant differences in concentrations among offices in smoking prohibited buildings and nonsmoking office areas in buildings where smoking was restricted to (1) rooms with local filtration, (2) areas with no local air treatment, (3) rooms with separate ventilation or (4) open workstations and enclosed offices. (See Ex. 3-1074) Further, investigators from Healthy Buildings International recently summarized results of their paper entitled "The Measurement of Environmental Tobacco Smoke in 585 Office

Environments." Using measured nicotine and particulate levels as markers for the presence of ETS, the investigators reported "spillover" of ETS in only 4% of the offices investigated. (See Ex. 3-1074)

The data, therefore, support the contention that designated smoking areas operated under negative pressure can essentially eliminate ETS exposure in nonsmoking areas, even under conditions of recirculation.

III. OSHA HAS NEITHER CONDUCTED A PROPER REGULATORY IMPACT ANALYSIS NOR ADEQUATELY EVALUATED THE ECONOMIC IMPACT OF THE PROPOSED STANDARD

A. Assessing the costs of the standard

In an effort to increase accountability for regulatory action, federal agencies must prepare a preliminary and final Regulatory Impact Analysis (RIA) for each major rule. To the extent permitted by law, each RIA must contain:

> (2) a description of potential costs of the rule, including any adverse effects that cannot be quantified in monetary terms, and identification of those likely to bear costs;

(Executive Order 12291)

While the Supreme Court in Cotton Dust supra, held that OSHA may not conduct a formal cost benefit analysis in its standard setting procedure, the Agency must still comply with the Executive Order. In conducting the RIA, the Agency must assess the costs and adverse effects of the proposed standard. In the instant situation, OSHA has developed no information whatsoever on demolition, design, installation, or material costs for separately ventilated designated smoking areas; revenue or sales losses for businesses affected by patron desertion; Agency enforcement expenses; or the loss of state revenues due to reduced sales of taxable tobacco products.

Rather, OSHA merely asserts that the average cost of "retrofitting the HVAC system [for a designated smoking area that complies with the proposed standard] ranges from $4,000 for a 150 square feet room . . . to $25,000 for 1,000 square feet . . . . " (59 FR at 16018) Moreover, in making its industry wide cost projections, OSHA assumed that "50 percent of all eating and drinking places and hotels and other lodging places may provide separate designated smoking areas." (Id.) However, the Agency's own interpretation of the standard prohibits the performance of work in a designated smoking area. Following this interpretation, OSHA has effectively banned smoking in such establishments as restaurants and hotels as no restaurant or hotel could require an employee to work in an area designated for smoking. In sum, OSHA failed to consider monetary costs and other adverse effects the standard will have on these businesses.

B. Economic feasibility

Section 6(b)(5) of the OSH Act provides that a standard developed to address with employee exposure to toxic materials or harmful physical agents shall adequately ensure, to the extent feasible, that no material health impairment will ensue. Although OSH Act §3(8), the definition of an occupational safety and health standard, by its terms does not require a feasibility analysis, the Supreme Court stated that "any standard that [is] not economically or technologically feasible would a fortiori not be 'reasonably

necessary or appropriate' under [federal law]." (American Textile Manufacturers, 452 U.S. 490, 513 n. 31 ("Cotton Dust"))

An "economic feasibility" evaluation does not require a cost-benefit analysis. (Cotton Dust, 452 U.S. at 507, n. 26; 43 FR at 27379) However, Philip Morris submits that OSHA "must . . . provide a reasonable assessment of the likely range of costs of its standard, and the likely effects of those costs on the industry," United Steelworkers, 467 F.2d at 1266, so as to "demonstrate a reasonable likelihood that these costs will not threaten the existence or competitive structure of an industry, even if it does portend disaster for some marginal firms." (467 F.2d at 1272) OSHA must support an economic evaluation with substantial evidence, and must address the impact of the regulation on all affected industries. (967 F.2d at 301 n. 160. Accord, AFL-CIO v. OSHA, 965 F.2d 962, 982 (11th Cir. 1992) (the determination of economic feasibility is governed by the same principle as technological feasibility; it must be supported by substantial evidence and OSHA must demonstrate its applicability to the affected industries); ASARCO, Inc. v. OSHA, 746 F.2d 483, 500 (9th Cir. 1984))

OSHA may not conduct the economic feasibility analysis by broadly grouping together various components of an industry "sector," (defined by the two-digit SIC code) absent a specific explanation of why such overbroad generalities are appropriate. (AFL-CIO v. OSHA, 965 F.2d at 982) As the Eleventh Circuit

recognized, reliance on "tools [such] as average estimates of cost can be extremely misleading in assessing the impact of particular standards on individual industries. Analyzing the economic impact for an entire [industrial] sector could conceal particular industries laboring under special disabilities [which are] likely to fail as a result of enforcement." (965 F.2d at 982)

In contrast, OSHA properly analyzed economic feasibility where it quantified the specific costs for each separate noise standard regulatory requirement to reach a total cost per industry worker. (Forging Industry Association v. Secretary of Labor, 773 F.2d 1436 (4th Cir. 1985)) The Fourth Circuit commented: "With respect to each component, [OSHA] exhaustively analyzed and evaluated data submitted by the industry and the scientific community and fully explained the basis of its computations." (773 F.2d at 1453) The final economic analysis in Forging Industry was based on quantified costs of audiometric testing, total number of workers, decreased production due to test requirements, test fees, time lost because of physician referrals, and implementation costs, such as test booth, audiometer, and accessory expenses, operator certification costs and equipment calibration fees. (773 F.2d at 1453-54) The court found that substantial evidence adequately supported OSHA's economic feasibility estimate.

Philip Morris submits that "the undisputed principle that feasibility is to be tested industry-by-industry demands that OSHA

examine the [economic] feasibility of each industry individually." (965 F.2d at 980 (citations omitted)) Nevertheless, OSHA conducted its economic feasibility analysis at the two-digit SIC code level in defiance of the Eleventh Circuit's finding in Air Contaminants. Further, OSHA estimated only the compliance cost as a percentage of revenue and profit. It failed to consider the other economic effects of implementing the proposed standard. The only explanation OSHA offered for its inadequate analysis was that "[t]his has been [its] procedure for doing regulatory impact analyses. . . ." (59 FR at 16108) By grouping affected industries into such broad classifications and failing to consider the economic impact of the standard on an industry by industry basis, OSHA failed to establish that its proposed standard is economically feasible.

IV. PRACTICAL CONSIDERATIONS POSED BY THE SCOPE OF THE PROPOSED STANDARD

A. The proposed standard's smoking restrictions apply in situations over which OSHA has no rational basis to regulate, and do so in a manner never before used by the agency

The proposed standard's smoking restrictions apply to "all indoor or enclosed workplaces under OSHA jurisdiction." (29 C.F.R. 1910.1033(a)(2)) The OSH Act, however, does not define "workplace." Instead, the Act uses the definitions of "employers" and "employees" to define OSHA's jurisdiction. The Act defines "employer" as "a person engaged in a business affecting commerce who has employees, but does not include the United States or any State or political subdivision of a State." (29 U.S.C. § 652(5)) "Employees" are defined as "an employee of an employer who is employed in a business of his employer which affects commerce." (29 U.S.C. § 652(6))

The approach taken in this proposal to apply the standard to workplaces as opposed to employee exposures is unique in OSHA regulatory history of toxic substances. Rather than basing regulatory requirements on employee exposure, as every standard OSHA ever promulgated addressing a toxic substance has done, the regulation actually uses enclosed work environments as a surrogate for employee exposure. Actual employee exposure is meaningless; it is the "room" that must be protected. As a result, the proposed regulation makes no distinction between one cigarette smoked in an enclosed football stadium over one hundred yards from the nearest

employee and 10 cigarettes smoked simultaneously in a small unventilated conference room. In the eyes of the regulation, both situations pose an equal risk to an employee. This causes the proposed regulation to apply in situations where clearly no rational justification for regulation exists.

In addition, the concept of eliminating exposure in places as opposed to addressing exposure to employees also becomes unworkable when the concept of time is introduced. Because it is the act of smoking in the enclosed workplace that is regulated, not the level of employee exposure to tobacco smoke, time becomes meaningless. For example, the regulation could have the effect of banning smoking in an office which did not meet the specifications of a designated area. What would an employer's obligation be if a nonemployee had smoked one cigarette in that office, when no employees were present, one minute before an employee entered? One hour before? The day before? On the face of the regulation the employer could not require the employee to enter that office, even if the one cigarette had been smoked months or years before. Language in the preamble would go even further and prohibit the employee from entering the office even if he or she wanted to. Obviously, this makes no sense.

The following examples illustrate the problems that arise from the application of the regulation consistent with the preamble's interpretation and the proposed regulatory text.

1. Private residences

The proposed standard covers private residences, raising the prospect of OSHA citing homeowners and home service companies for exposing their employees to ETS. The IAQ portions of the proposed standard apply only to non-industrial workplaces, which are defined to expressly exclude manufacturing or production facilities, residences, vehicles and agricultural operations; however, the ETS portion of the proposed standard applies to "all indoor or enclosed workplaces," which clearly implies that all of these workplaces would be covered. Where a housekeeper, nanny, nurse, repair person, delivery person, or any other employee performs work in a private residence, the proposed standard would require the employer (i.e., either the homeowner or home service contractor) to restrict smoking in the home to a designated smoking room meeting the specifications of the standard. This room must not be where any work is to be performed. Homeowners who cannot afford to install such designated smoking rooms would be required to prohibit smoking in their homes entirely.

Because the proposed standard does not specify <u>when</u> smoking must be prohibited, and because the preamble's mandate is that an employee cannot be required to work where "contamination" from ETS is present (59 FR 16001), every home that may at some time or another be a workplace would have to be smoke-free, even though

no employees may have been present in the home for days, weeks, months or years.

Similarly, the proposed standard does not specify when "no smoking" signs must be posted at the entrances of workplaces, including homes. (29 C.F.R. § 1910.1033(e)(1)(vi)) Thus, the proposed standard would seem to require that such signs be posted continually, presumably at all entrances to private residences.

2. Hotels and motels

The proposed standard would also cover sleeping rooms in hotels and motels because service, custodial, and maintenance staff are regularly employed in such rooms. Like the restriction on smoking in the home, the proposed standard would prohibit smoking in hotel rooms at all times, not just while employees are working in the room, because of "contamination" due to ETS. Even though the proposed standard permits custodial and maintenance employees to work in unoccupied designated smoking areas (29 C.F.R. § 1910.1033(e)(1)(iii)), smoking would still be prohibited in sleeping rooms of hotels and motels because other employees, such as bellhops and room service employees, are required to enter the room.

3. Restaurants, bars, and other service establishments

The proposed standard would also cover restaurants and bars, bowling alleys, and other service establishments. Because designated smoking areas in such establishments must not be work areas, customers could not smoke at tables or bars where waiters and bartenders work. This restriction would effectively ban smoking by patrons of restaurants and bars entirely, as well as at catered private parties, bowling alleys, pool halls, bingo halls, casinos, etc. Such an effect would undoubtedly have a significant negative economic impact. This same restriction on customer smoking would also apply to retail establishments, shopping malls, indoor sports arenas, and any other enclosed public area where there is just one person who fits the broad definition of "employee."

4. Nursing homes

Nursing homes and long term health care facilities will be faced with unique hardship if OSHA promulgates the proposed standard. These facilities are required by the Social Security Act to recognize numerous "Residents' Rights." Among these rights, a resident has the right to "make choices about aspects of his or her life in the facility that are significant to the resident." (42 C.F.R. § 483.15(b)(3)) In its interpretive guidelines, the U.S. Department of Health and Human Services also states that any

nursing facility that chooses to prohibit smoking "must allow current residents who smoke to continue smoking in an area that maintains the quality of life for these residents." (Interpretive Guidelines, § 483.15(b)(3)) Although the guidelines allow nursing homes to designate outdoor areas for smoking, the facility must provide an alternative area on days when the weather could make it unsafe or unhealthy to smoke outside.

According to these guidelines, nursing homes are effectively foreclosed from prohibiting smoking. As a result, most, if not all, nursing homes are currently accommodating their residents who are smokers. The proposed standard would, in effect, require nursing homes to build designated smoking areas that meet the specifications of the standard.

However, nursing home employees would be prohibited from entering designated smoking areas at all times, except to perform custodial or maintenance work when smoking is not taking place. This means that a nurse would be legally prohibited from pushing a wheelchair-bound resident into the designated smoking area, and from rendering emergency medical treatment to a resident who might require treatment while in a designated smoking area, even when smoking is not taking place.

5. Vehicles

The proposed standard would prohibit employees from smoking in vehicles used in the course of employment, even with all of the windows open. Such vehicles, whether a company truck or an employee's personal car, are indoor workplaces and are thus covered by the smoking provisions of the proposed standard. Like the restriction on smoking in the home, the "letter" of the proposed standard prohibits smoking in all vehicles that have at some time or another been used in the course of employment. Smoking would therefore be prohibited in such vehicles at all times, not just during company business.

Also, if an employee's car has been used in the course of employment, he would be prohibited from smoking in that vehicle even while alone. To prohibit an employee from smoking while alone in his own vehicle is patently unreasonable.

6. International airline flights

Under the proposed standard, employers could not require their employees to take international airline flights because smoking is permitted on those flights. In addition, flight attendants could not be required to enter those cabins where smoking had occurred in order to serve passengers.

7. Tobacco shops

The proposed standard contains no exclusion for those shops engaged primarily in the sale of tobacco and tobacco-related accessories. Patrons of such shops are accustomed to sampling the various tobacco blends and products while on the premises in order to determine the blend or product they prefer. The proposed standard would require the shops to build designated smoking areas, separate from the shops' work areas, and would prohibit the shops' employees from serving customers in those areas.

8. Scientific research facilities

The proposed smoking restriction contains no exclusion for smoking that is necessary to scientific research. Under the current proposal, smoking experiments could not be conducted in designated smoking areas because laboratory employees could not work in those areas.

9. Arts and entertainment

The provisions in the proposed standard that prohibit smoking of tobacco products in any work area make no exceptions for the arts and entertainment industries. Thus, the proposed standard, as currently drafted, would ban the use of tobacco products in any movie scene. Likewise, a live theatrical

production could not permit the smoking of tobacco products by any character in the production. Similarly, a model could not be employed to pose for a photograph with a cigarette as this activity would not (and could not) be limited to a non-work area as required by the proposed standard.

B. The proposed standard's smoking restrictions improperly interfere with "personal choice"

The regulation of indoor smoking presents unique legal, policy and practical issues. Many of these issues stem from the concept of governmental involvement in "personal choice" issues. In its proposal, OSHA seeks to address a perceived harm from nonsmoker exposure to ETS. The Agency has not chosen to address the issue of the smoker's exposure to his or her own smoke, either from the act of smoking itself, or from the exposure to the smoke from other smokers in a designated area. Presumably, this choice was made in recognition that these activities fall within the personal choice of the employee. If OSHA were to determine that the personal choice to smoke was an appropriate subject for government regulation, then OSHA could, under exactly the same theory, ban an employee's consumption of red meat, salt, coffee, sugar substitutes, butter, or fried chicken.

The Agency offers no rationale for this distinction, and it appears to be a pure policy choice. Its implications are

important, however, because once such a policy choice is made, the remainder of the regulation must be consistent with that policy.

It is apparently the Agency's intent to require employers to prohibit employees from working in a designated smoking area. The preamble states that "no work of any kind shall be performed in a designated smoking area when smoking is taking place" (59 FR 16029), and that "[n]o employee can be required to work in an area where there will be contamination from ETS" (59 FR 16001) [emphasis added]. This position is not consistent with the proposed regulatory text, however, which provides that cleaning and maintenance can be conducted in such areas as long as no smoking is taking place at that time (29 CFR § 1910.1033(e)(1)(iii)) and that employees are not required to enter designated smoking areas in the performance of normal work activities. (29 CFR § 1910.1033(e)(1)(iv))

This distinction between the preamble and the regulatory text is important because, based upon the language in the regulatory text that no employee can be "required" to enter a designated area, a smoker could, for example, choose to read a work-related memorandum while in a designated area. A smoker could also choose to enter the designated area to discuss a work-related matter with a smoking co-worker. The language in the preamble, however, would dictate that the work-related reading of the memorandum or the discussion with the co-worker would be

prohibited. This would place OSHA in the absurd position of not only ensuring that no person smoke while not in a designated area, but that no person perform any work while in a designated area.[27/] The concept of OSHA issuing a citation to an employer for failing to ensure that no work was performed in a designated smoking area is mind-boggling.

Clearly, if the Agency incorporates provisions regarding indoor smoking in its final regulation, a common sense approach must be applied. As such, even under its proposed standard, the following situation would appear to be permissible, based upon the specific language of the proposed rule: If the employee desired, his or her private office could be a designated smoking area provided that no other employee was required to enter as part of normal work activities. This would not preclude any other employee from volunteering to enter the office/designated area, even though that employee might not be a smoker, as long as that other employee was not required to enter.

It would simply be illogical and unworkable to attempt to treat "smoking" employees and "nonsmoking" employees differently, because the difference in their status only occurs when the employee actually lights a cigarette or other tobacco product. In

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[27/]Interestingly, the Wage Hour Interpretive Bulletins, 29 CFR § 785.18, would require that employers compensate employees for this time in the designated smoking area, even though OSHA would prohibit any work from being performed.

fact, a "nonsmoking" employee can become a "smoking" employee, simply by declaring that he or she smokes. Accordingly, if a "smoking" employee can work in a designated area, so could a nonsmoking employee, if they chose to do so.

Given these clear practical problems with the application of the regulation as currently drafted, OSHA, if it determines that regulatory action is warranted at all, must look at other options. Two approaches deserve consideration. First, OSHA should consider a permissible exposure level for one or more of the constituents of ETS. This approach would address ETS exposure and could also be integrated with the agency's regulation of other indoor air constituents. For example, a permissible exposure level based upon carbon dioxide as a surrogate for both adequate ventilation and acceptable exposure levels of ETS would be consistent with OSHA's approach to every other airborne toxic substance OSHA has ever addressed. It would also not create an artificial differentiation between smoking and nonsmoking employees, because an employer would be required to ensure that no employee was exposed to levels of carbon dioxide over the PEL.

Another regulatory approach, which would recognize the problems inherent in the regulation of an issue involving personal choice, would be a regulation which simply prohibited an employer from requiring an employee to work in an area where smoking is currently taking place. This approach would be much easier to

enforce because the compliance officer would only have to determine whether an employee was required to work in an area at a time when anyone (employee or patron) was smoking. Either of these options would establish a much more workable regulatory scheme.

V. THE STANDARD IS UNCONSTITUTIONALLY VAGUE AND UNENFORCEABLE

Civil statutes and regulations, like the proposed IAQ standard, are subject to judicial review for vagueness. (Village of Hoffman Estates v. Flipside, 455 U.S. 489 (1982)) In cases involving regulations that impose penalties for violations, such as OSHA standards, courts consider whether the law is sufficiently definite to provide notice of the conduct it prohibits or requires and to guide those that must apply it. Recently, the Eleventh Circuit in Georgia Pacific Corp. v. O.S.H.R.C., No. 93-6503 (11th Cir., July 13, 1994), held that the OSHA standard pertaining to the operation of forklifts was unconstitutionally void as applied. The standard provided: "If the load being carried obstructs forward view, the driver shall be required to travel with the load trailing." (29 C.F.R. § 1910.178(n)(4)) During an enforcement proceeding, OSHA and the cited party suggested several interpretations of the words "obstructs forward view." In reviewing the standard for vagueness, the court asserted: "Like other statutes and regulations which allow monetary penalties against those who violate them, an occupational safety and health standard must give an employer fair warning of the conduct it prohibits or requires." (Id., citing Diamond Roofing v. O.S.H.R.C., 528 F.2d 645 (5th Cir. 1976)) Given the varying interpretations of the standard's requirements, the court found "that where the Secretary is unable to settle upon a single definition of a critical term or phrase of its own regulation, that

the regulation is unconstitutionally vague as applied for failing to give sufficient guidance to those who enforce OSHA penalties, to those subject to civil penalties, or to those courts who may be charged to interpret and apply the standards." (Georgia Pacific at LEXIS 18)

The proposed standard is ambiguous with respect to both the physical and temporal scope and application of its provisions. First, it is so vague with respect to the types of enclosed workplaces it covers that its adoption and enforcement by OSHA deprives interested parties of a reasonable opportunity to determine which of their facilities, operations, homes, vehicles, etc. are covered by the standard and to comply accordingly. For example, although the standard does not specifically mention that it covers private residences, its general coverage of indoor work areas implicitly includes homes where an employee works. Where a housekeeper, repair person, delivery person, home office worker, family member or any other employee performs work in a private residence, the "letter" of the standard would require the employer (i.e., the home owner or home service contractor) to restrict smoking in the home to a designated area meeting the costly specifications of the standard. In effect, this interpretation of the standard would prohibit home owners from smoking in their own homes. It also appears from the language of the standard that such home owners would be prohibited from smoking at any time, not just while an employee is working in the home.

Second, the proposed standard requires that "no smoking" signs be posted at every entrance to all enclosed workplaces covered by the standard. It appears that this requirement applies to every home nationwide, almost all of which at some time or another have employed someone, and to every vehicle used in the course of employment. The standard is unclear regarding whether it requires the posting of such signs continually, or only when a employee is working in the home or using the vehicle in the course of employment.

Finally, the standard is so vague in scope and application that it fails to provide legally fixed standards and adequate guidelines for OSHA, the courts, and others whose obligation it is to enforce, apply and administer the standard.

The lack of legally fixed enforcement standards renders enforcement impossible. Potentially, the proposed standard would regulate smoking in homes and businesses nationwide. As it stands, OSHA has difficulty monitoring existing workplaces for compliance with current standards. The enforcement of a standard as broad and vague as the proposed IAQ standard would require a virtual police state.

Thus, the proposed standard is vague to the point of unenforceability. Adoption and enforcement of such a vague

standard would deprive employers of their due process rights in violation of the Fifth Amendment of the United States Constitution.

VI. THE STANDARD IS UNCONSTITUTIONALLY OVERBROAD

A law that properly regulates certain activity but also infringes upon constitutionally protected rights is unconstitutionally overbroad. (Village of Hoffman Estates v. Flipside, 455 U.S. 489 (1982)) Application of the proposed standard to homes, sleeping rooms in hotels and motels, and private vehicles unnecessarily and unreasonably infringes upon the federal constitutional right to privacy that arises as a "penumbra" of the First, Fourth, Fifth, Ninth and Fourteenth Amendments to the United States Constitution.

In Stanley v. Georgia, 394 U.S. 557 (1969), the U.S. Supreme Court recognized a zone of privacy in one's home. From the First, Fourth, Fifth, Ninth, and Fourteenth Amendments ascends a penumbra of rights creating a constitutionally protected expectation of privacy in one's home. (Id. at 564-65) The Court held, "The right to be free except in limited circumstances from unwanted governmental intrusions into one's privacy is fundamental." (Id. at 566)

The Supreme Court has a long history of affording constitutional protection to activities conducted within the confines of the home. The Fourth and Fifth Amendments run a parallel course as protection against government invasions "of the sanctity of a man's home and the privacy of his life." Boyd v.

United States, 116 U.S. 616, 630 (1986) (Court stated that the "essence of a Fourth Amendment violation is not the breaking of a person's doors, and the rummaging of his drawers, but rather is the indefeasible right of personal security, liberty and private property"); see also, Griswold v. Connecticut, 381 U.S. 479, 485 (1965) (Court noted that intrusion into one's home to enforce an anti-contraception statute is constitutionally intolerable); Stanley v. Georgia, supra (Court stated that the First and Fourteenth Amendments protect possession of obscene materials in a private home); Payton v. New York, 445 U.S. 573, 603 (1980) (Court overturned conviction that resulted from warrantless and non-consensual entry into the defendant's home to effect arrest). Moreover, government intrusion into a person's private home either physically or by regulation merits some constitutional protection. See, Moore v. City of East Cleveland, 431 U.S. 494, 500 (1977) (Court held that a local ordinance that restricted living arrangements within private homes unconstitutional). Even actions that are clearly not protected constitutionally, such as publicly showing obscene films to consenting adults, Paris Adult Theaters I v. Slaton, 713 U.S. 49, 66 (1973), are given a heightened level of protection under the federal Constitution when done in the privacy of one's own home. Stanley v. Georgia, supra, (finding that although there is no per se protection for possession of obscene material, possession within the home receives constitutional protection); see also, Katz v. United States, 389 U.S. 347, 361 (1967) (Court stated that the expectation of privacy in one's own

home is high and activities conducted in the privacy of home deserve more constitutional protection than activities and statements exposed to the public). These cases indicate that specific activities, like smoking, while not amounting to fundamental rights, may still receive constitutional protection when conducted in the home and thereby demand increased scrutiny.

In addition to infringing upon employers' right to privacy, the proposed standard also unnecessarily and unreasonably infringes upon their right to freedom of speech, in violation of the First Amendment to the United States Constitution.

As explained above, the standard's broad application may require the posting of "no smoking" signs at every entrance to nearly every home nationwide and on the doors of every vehicle used in the course of employment. The standard also could be interpreted to require the posting of such signs continually, not just when an employee is working in the home or using the vehicle in the course of employment. Aside from the proposed rule's unreasonable and unnecessary scope, requiring "no smoking" signs to be posted year-round on private residences and private vehicles, just because those areas are at times a workplace, is unconstitutional forced speech.

The United States Supreme Court has held on numerous occasions that forced speech (e.g., government mandated posting of

signs) violates the First Amendment of the Constitution. Wooley v. Maynard, 430 U.S. 705 (1977); see also, Pacific Gas & Electric v. Public Utilities Commission of California, 475 U.S. 1 (1986); Harper & Row Publishers, Inc. v. Nation Enterprises, 471 U.S. 524 (1985); Miami Herald Publishing Co. v. Tornillo, 418 U.S. 241 (1974).

Because the proposed standard unreasonably and unnecessarily infringes upon employers' right to privacy and freedom of speech, it is unconstitutionally overbroad.[28/]

[28/]The constitutional nature of the rights the Standard infringes upon also calls for stricter scrutiny for vagueness. Village of Hoffman Estates, 455 U.S. at 499.

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SECTION III

WORKPLACE IAQ AND SIGNIFICANT RISK:

OSHA'S PROPOSED RULE

AND

SEPARATE RULEMAKING FOR ETS

WORKPLACE IAQ AND SIGNIFICANT RISK: OSHA'S PROPOSED RULE AND SEPARATE RULEMAKING FOR ETS

OSHA'S PROPOSED RULE ON IAQ RECOGNIZES THE IMPORTANCE OF THE PROPER DESIGN, MAINTENANCE, AND OPERATION OF A BUILDING'S HEATING, VENTILATION AND AIR CONDITIONING (HVAC) SYSTEM; THE PROPOSED RULE, HOWEVER, FALLS SHORT OF PROVIDING A COMPREHENSIVE APPROACH TO WORKPLACE IAQ; OSHA FAILS TO ADEQUATELY SUPPORT ITS OWN PROPOSED RULE AND FAILS TO DEMONSTRATE A SIGNIFICANT RISK OF MATERIAL IMPAIRMENT FROM IAQ

OSHA's Proposed Rule on indoor air quality (59 FR 15968) is based upon the following contentions:

(1) **Indoor air quality problems are most often associated with inadequate ventilation system performance.** The Proposed Rule states: "A common theme that runs through the literature and the OSHA docket indicates that the principal factor associated with indoor air quality complaints is inadequate ventilation" (59 FR 16010);

(2) **Complaints regarding unacceptable indoor air quality (usually associated with symptoms of irritation, annoyance and discomfort) cannot, as a rule, be traced directly to specific airborne substances or to specific levels of exposure.** The Proposed Rule states: "air quality complaints usually have some basis, although they are often difficult to assess with

specificity . . . [complaints] are not easily traced to a specific substance, but are perceived as resulting from some unidentified contaminant or combination of contaminants" (59 FR 15970); and

(3) **Remediation and mitigation of poor indoor air quality, and the attainment and maintenance of acceptable indoor air quality, depend upon the correct design, operation and maintenance of a buildings' ventilation system.** The Proposed Rule states: (a) "Symptoms . . . may be reduced or eliminated by modifying the ventilation system" (59 FR 15970); (b) "The outside air ventilation rate of the building affects indoor air quality. It determines the extent to which contaminants are diluted and removed from the indoor environment" (59 FR 16003); (c) Sick-building investigations report "the lack of outside ventilation air resulting from operational or maintenance deficiencies as one of the causes of IAQ complaints. Many of the studies include abatement recommendations to ventilate with outside air as feasible per the original design intent . . . [R]esearch projects . . . also support the case for ventilating buildings with at least the recommended minimum of outside air." (59 FR 16027)

Inadequate ventilation is associated with approximately one-half of all complaints in sick-building investigations

A number of summaries and databases of "sick-building" investigations were submitted to the docket on OSHA's 1991 Request for Information on Indoor Air Quality (RFI). With one exception, the Proposed Rule fails to reference or discuss any of them. One of the most notable compilations of sick-building investigations was undertaken by NIOSH, referenced in the OSHA Proposed Rule at 59 FR 16003 and 16010. Inadequate ventilation was identified as a primary problem in fifty-two percent (52%) of the 484 building investigations in the NIOSH database.[1,2] Inside contamination was identifiable in only fifteen percent (15%) of all buildings; microbiological contaminants were identified in five percent (5%).

Other building investigation databases that were neither referenced nor discussed in the Proposed Rule report similar results. For example, a database of 1,362 building investigations has been compiled by Health and Welfare Canada.[3,4] Inadequate ventilation was identified as the primary problem in fifty-two percent (52%) of the buildings investigated. Specific indoor air contaminants were identified in only twelve percent (12%) of the investigations. (Exs. 3-1073, 3-1074)

Public Works Canada, another Canadian federal agency, investigated 30 buildings for IAQ complaints between 1987 and 1990.[5] Ventilation-related problems were reported in one-half of the buildings. (Ex. 3-1073)

TDSA Ltd. have compiled data on 408 building investigations conducted in the U.S. and Canada. (Ex. 3-1073) The results have been analyzed and computerized in what is called the "Building Performance Database." Ventilation-related inadequacies were directly associated with IAQ complaints in forty-nine percent of the buildings catalogued in the Database.[6]

A private, U.S.-based IAQ monitoring firm conducted 412 building investigations from 1981 through 1988. (Ex. 3-1053) Ventilation problems were associated with complaints in sixty-two percent of the buildings investigated; bacterial and fungal contamination was reported in nearly a third of all buildings investigated.

A 1989 report by Dr. James Woods assessed 30 cases of "problem buildings" investigated by the Honeywell Corporation since 1986. Woods' research indicates that 75 percent of the buildings investigated had inadequate outdoor supply air intake. Similarly, 75 percent of the buildings exhibited inadequate air distribution

to occupied spaces, and 65 percent of the buildings suffered from inadequate HVAC maintenance.[7] (Exs. 3-745, 3-1074)

Kim (1990) summarized 105 investigations of problem buildings undertaken by Clayton Environmental Consultants, an IAQ monitoring firm in the U.S.[8] (Ex. 3-505), and wrote:

> In a survey of 105 buildings, Clayton found that 53 percent had [HVAC] maintenance problems, 49 percent had operational problems (such as improper handling of control equipment), and 33 percent had design problems. Mechanical engineers evaluated the HVAC systems in 70 of the buildings, in which they found that 75 percent had maintenance problems, 70 percent had operational problems, and 47 percent had design problems. Of the 105 buildings, 95 were sampled for contaminants. Of these, 28 were found to have problem levels of microbial contaminants, 26 had volatile organic compounds and 13 combustion products. (Ex. 3-1074)

Freund, et al., from the New Jersey Department of Health, evaluated 221 complaint buildings in that state and reported that 43 percent were associated with inadequate ventilation.[9] (Ex. 3-1053)

The Oregon Department of Resources submitted results of 36 state office building investigations to the OSHA RFI docket. (Ex. 3-1157) The respondents observed:

> Thirty-six state office buildings were examined for IAQ problems. Significant

> problems were found in 16 buildings. Many of the problems were associated with inadequate ventilation and high levels of carbon dioxide . . . [S]tudies in Oregon have shown that when outside make-up air falls below 15 cfm, complaints increase.

In a submission to OSHA by the Local 12/Occupational Illness Support Group of the U.S. Department of Labor, the authors report that:

> Inadequate ventilation is the primary cause of most of the indoor air quality problems . . . . This is a result of the Department's inability to maintain adequate amounts of outside air. (Ex. 3-1017)

Thus, published data from a number of building investigation databases submitted to OSHA reveal that deficiencies in ventilation have been directly related to IAQ complaints in approximately one-half or more of all reported cases. HVAC-related problems have been associated with complaints in as many as 75 percent of the sick-building investigations discussed above.

IAQ complaints generally cannot be associated with specific substances

Although the Proposed Rule correctly contends that correlations between specific complaints and specific substance exposures in indoor air quality investigations are rare, it fails to cite relevant support from materials submitted to OSHA in the RFI docket. (59 FR 15969) A number of comments submitted to the

docket substantiate that claim. For example, the Atlantic Richfield Company (ARCO) reports that, in their experience, "when monitoring has been conducted, hazardous contaminants have either not been detected, or they are present in concentrations far below those known to present health hazards." (Ex. 3-448)

The submission from Organization Resources Counselors states that:

> Companies report monitoring for formaldehyde, total and respirable particulates, total organics. . . . In almost all cases where monitoring was done for specific contaminants, results were either below the level of detection, or were below OSHA Permissible Exposure Limits. (Ex. 3-1084)

Similarly, CanTox, Inc. reports that:

> More than half (63 percent) of the compounds detected in indoor air could not be attributed to a definite source. The largest group with known sources were found to have multiple sources and their presence could not be exclusively attributed to one specific source. This clearly has significant implications with respect to attempting to use source control to maintain indoor air quality. (Ex. 3-1180)

The American Federation of Government Employees (AFGE) of the AFL-CIO reports that "unfortunately, despite the substantial evidence linking poor indoor air quality to AFGE members' adverse health effects, AFGE is unable to obtain the data needed to make

the causal connection between specific contaminants and those adverse health effects." (Ex. 3-529)

United Technologies reports that, "based on our experience in occupational settings and knowledge of the professional literature, there are only very weak data that directly relate specific chemicals to IAQ." (Ex. 3-651)

TDSA Ltd., after citing four studies, reports that:

> The correlation between symptoms presented in IAQ complaints and causative agents is weak because exposure to many different types of contaminants in indoor air, originating from both indoor and outdoor sources, has been shown to result in similar health and comfort complaints. The presence of pollutants in indoor air, combined with thermal comfort parameters of temperature and humidity, and other building characteristics, such as ventilation, lighting, noise and occupant density and activities, make it difficult to isolate the causative agent in IAQ-related health and comfort complaints. (Ex. 3-1073)

The American Industrial Hygiene Association (AIHA) notes that, based on their experience:

> In most IAQ complaints symptoms are non-specific and could be caused by a variety of factors. Correlations can be found where there is a consistent spacial and temporal relationship and the complaint can be resolved by changing the building condition. Biological contamination may sometimes be distinguished by a pattern of allergy symptoms. (Ex. 3-735)

The Building Performance Database compiled by TDSA Ltd. provides data on airborne substance monitoring from over 200 sick-building investigations. Average recorded levels of carbon dioxide, carbon monoxide, respirable suspended particles, formaldehyde, airborne fungi and bacteria, nicotine, temperature and humidity are all within parameters of "acceptable" exposure. Nevertheless, the buildings from which the monitoring results arose were deemed "sick." The authors conclude: "In general, IAQ investigations of white collar workplaces have found indoor concentrations of measured substances far below occupational exposure levels." (Ex. 3-1073)

The sick-building databases submitted to the OSHA RFI Docket indicate that ETS is associated with complaints in only two to five percent of all investigations; the data do not support OSHA's attempt to separate ETS from general IAQ issues

OSHA's Proposed Rule conspicuously omits any discussion of ETS within the context of its discussion on IAQ and the identification of specific causes in sick-building investigations. The major databases on sick-building syndrome submitted to the OSHA RFI docket indicate that tobacco smoke is rarely the underlying cause of complaints about poor indoor air quality.[1,3,6] For example, in HBI's database of 412 sick-buildings, ETS was reported to be a significant contributor to complaints in only 3 percent of all

buildings investigated. (Ex. 3-1053) In the sick-building database compiled by TDSA Ltd., smoking was implicated as a major contributor to complaints in only 12 of 408 (less than 3 percent) of the buildings surveyed.[6] (Ex. 3-1073) NIOSH investigated more than 200 sick-buildings through 1984 and reported that tobacco smoke was a source of claimed discomfort in only 2 percent of the buildings investigated.[1] (Ex. 3-1074) In a summary of 94 building studies by government investigators from Health and Welfare Canada, complaints were attributable to indoor constituents such as photocopy machine emissions and ETS in only 5 percent of the buildings investigated.[3] (Ex. 3-1074)

Professor Alan Hedge of Columbia University submitted the results of his study on 4,479 office workers from 27 air-conditioned offices to the RFI docket. (Ex. 3-955) Hedge examined the potential impact of smoking and smoking policies on reports of sick-building syndrome. He reported that sick-building complaints could not be correlated with levels of ETS constituents in the indoor air, and that workers in smoking-prohibited buildings, on average, reported more symptoms than workers in buildings with restricted smoking policies.

One submission to the OSHA docket thus concluded: "Removing the smoker entirely, then, would not affect health and

comfort problems in 95 to 98 percent of sick-buildings." (Ex. 3-1073)

Ventilation: the mitigation procedure for poor IAQ

The data on ETS discussed above are derived from the same databases that support OSHA's contention regarding the primary cause of complaints about indoor air quality, namely, inadequate ventilation. The Proposed Rule correctly recognizes that the appropriate mitigation procedure for complaints focuses on ventilation, and, specifically, on adequate supply air, its distribution to occupied spaces, and the proper maintenance of HVAC systems. The RFI docket is replete with such recommendations. For example, the New York State Building and Construction Trades Council reports that, based on their experience, "the average office setting exposes workers to contaminants from machines, carpets, paints, glues, and fungi. These contaminants mix with the air the workers breathe on a daily basis and affect a person's well-being. Proper ventilation has been shown to provide a proven antidote to these problems." (Ex. 3-732)

IAQ investigators from AFSCME Local 12 from the University of Iowa Employees Union report that "we have found that insufficient fresh air flow is most often the cause of a number of

symptoms, including coughing, skin and eye irritations, headaches and upper respiratory infections . . . [I]ndoor air quality problems can be treated with little effort and expense by improving or upgrading inadequate ventilation systems to increase fresh air flow in the workplace." (Ex. 3-1171)

Investigators from CanTox Inc. agree: "[C]ontrol of the air exchange rate (i.e., ventilation) of a building is probably the most important and practical mediation practice for management of indoor air quality." (Ex. 3-1180)

The State of New Jersey Health Department's investigation of 221 complaint buildings revealed that over 43 percent of all cases involved inadequate ventilation.[9] In cases where abatement recommendations were made, the recommended strategy consisted of increased maintenance, repair, adjustment or redesign of the HVAC system. Eighty-four percent of the cases where such abatement steps were implemented reported the elimination of complaints. (Ex. 3-1053)

Indoor air quality in schools has recently become an issue of considerable concern. In 1989, Helsing and co-workers reported the results of an IAQ investigation in a school.[10] They reported that "there was an insufficient fresh air supply to some classrooms and a large percentage of students exhibited classic

symptoms of sick-building syndrome, i.e., headache, eye burning, fatigue." Similarly, investigations by Hanssen (1987)[11] and Beller (1989)[12] reported that low air exchange rates in combination with installation of new building materials were the main cause of complaints in the schools that they investigated. Helsing, et al. concluded: "Correcting the ventilation problems resulted in reduction of symptoms to a level approximately equal to that of students in other schools in the county."[10] (Ex. 3-1074)

In 1989, Collett and Sterling examined the effect of ventilation retrofits on perceived health and comfort complaints by building occupants. For the buildings in which major retrofits were undertaken, perceptions of indoor air quality improved in seven of nine categories surveyed. (Ex. 3-1073)

Similarly, health officials who investigated and compiled the Canadian sick-building database (Health and Welfare Canada) observed that recommendations for improvements in ventilation and thermal comfort had been made in 60 percent of the 1,400 buildings investigated, while control of specific pollutants was recommended in only 20 percent of all cases.[13] (Ex. 3-1074)

In 1984, a committee on sick-building syndrome from the WHO's Europe Working Group on Indoor Air Research concluded that an increase in outdoor air supply rates, together with improvements in

air distribution, had satisfactory results in remediating sick-building syndrome. (Ex. 3-188)

The Ohio Civil Service Employees Association, after reviewing three indoor air quality incidents in Ohio, endorsed the ventilation approach "as a vital component in solving and preventing persistent health and safety problems related to indoor air in the workplace." (Ex. 3-398)

Steven B. Hayward of the State of California Department of Health Services recommends adoption of a minimum ventilation standard similar to that currently in effect as a Cal/OSHA regulation. The Cal/OSHA standard requires that a minimum supply of outdoor air specified in the State Building Standard Code be provided (15 cfm/m^3), and that the HVAC system be operated continuously, inspected regularly, and properly maintained. (Ex. 3-17)

Respondents to the RFI also observe that adequate ventilation is the preferred method for controlling exposures to radon, VOCs, CO, CO_2, bioaerosols and ETS. (Exs. 3-500, 3-61, 3-1053) Indeed, the U.S. Department of Labor's own Occupational Illness Support Group states that ETS and radon "have seldom been the source of employee complaints of poor indoor air quality. With properly designed and properly operated ventilation systems,

exposure to radon and passive smoke can be minimized." (Ex. 3-1017)

OSHA's Proposed Rule fails to address other environmental variables unrelated to IAQ that play a role in worker complaints about health and comfort

The Proposed Rule dismisses the possibility that factors unrelated to IAQ, e.g., temperature, lighting, stress, workload, etc., may play a role in worker perception about IAQ. OSHA discounts the potential psychological element in worker complaints by arguing that complaints "are unlikely to be due to mass psychogenic illness." (59 FR 15970) OSHA misses the point. A number of studies in the published literature that were submitted to the RFI docket indicate that lighting, temperature, humidity, job satisfaction, job stress and ergonomics are factors that influence worker perceptions about IAQ. (Ex. 3-1073) For example, in a SBS study in Denmark, Skov and colleagues examined sick-building syndrome reports among 4,369 office workers.[14] Their research indicated that indoor climate perception was strongly related to the prevalence of SBS symptoms. Lifestyle factors were only weakly associated with the reporting of symptoms. (Ex. 3-1074)

A 1991 report by Hawkins and Wang ranked a number of variables related to self-reported symptoms of sick-building syndrome.[15] Those variables included: "humidity," "satisfaction

with work," "active smoking," "gender," "exposure to ETS," "office light," and "doing professional work." They concluded:

> Building Sickness Score was associated with many factors. Sick building syndrome symptoms are influenced by multiple variables of which the environmental factor of humidity and the psychological factors of work, sex, and occupation are important. (Ex. 3-1074)

Based on one of his own studies, Hedge reports that although ventilation has an important effect on indoor air quality, reports from workers in 46 office buildings in the United Kingdom indicate that complaints are even more strongly influenced by a number of personal and occupational factors such as gender, job stress, job satisfaction and computer use. (Ex. 3-955)

The American Industrial Hygiene Association (AIHA) reports that "psychosocial and physical stresses are certainly potential causes of some IAQ complaints and should always be considered in any investigation." This opinion is based on the experience of AIHA members. (Ex. 3-735)

Citing two studies, Eagle Environmental Health reports that "thermal discomfort, unpleasant odors, lack of air movement, insufficient lighting, and excessive noise are also indicated in IAQ investigations. Job-related stress many also manifest itself in IAQ complaints." (Ex. 3-500)

The National Energy Management Institute (NEMI) reports:

> The NEMI experience has revealed that there are a variety of factors which can interact to cause a worker to display indoor environmental health-related problems. These factors may actually be the primary cause or may exacerbate an IAQ problem condition. Temperature, temperature change, humidity, air velocity, light levels, noise as well as psycho-social factors should always be considered in presenting and investigating IAQ problems. (Ex. 3-1183)

Even the U.S. EPA recognizes that factors related to SBS are multi-factorial, involving combined environmental and psycho-social stressors. (Ex. 3-1075, Attachment H) Citing the World Health Organization (1986), the EPA notes:

> Buildings at highest risk [of SBS] appear to be new or recently remodeled buildings with tight envelopes, especially those with large ventilation systems that depend on limited fresh air sources. Improper ventilation, thermal conditions, and occupant lack of control over climatic and working conditions are other factors that may increase the likelihood of a building being linked to sick-building syndrome. (Ex. 3-1075, Attachment E)

A NIOSH psychologist, Dr. Michael Colligan, has offered an explanation for the role of such factors in perceptions about IAQ.[16] (Ex. 3-1074) He writes:

> It appears then, that the individual is sensitive to fluctuations in the functioning of the autonomic nervous system. When perceived changes in his subjective state are understandable, e.g., 'I have an allergy,'

> 'I've been under a lot of pressure to met a deadline,' 'I'm worried about my teenager,' an individual can initiate various coping strategies to deal with the causes. When the origins of the experienced distress are vague or unclear, however, an individual starts searching around for salient cues. If the environment provides a plausible cause in the form of a pungent odor or dense, stuffy air, then an individual can conclude, rightly or wrongly, that the poor quality of the environment is responsible for his physical and psychological discomfort. Notice that this process can occur independently of any specific toxic effects the environment might have on the individual and irrespective of the 'real' cause of the autonomic arousal. All that is required is that individual experience autonomic arousal in response to a subtle or unidentified stressor or combination of stressors. Cues provided by the environment in the form of noxious odors, visually detectable particulates or dust, or humid, stuffy air, may suggest to an individual that his discomfort is a toxic response to an airborne pollutant. That environment then becomes a source of threat to the individual, which in turn may generate more autonomic arousal and anxiety.

Dr. Colligan's observations provide an understanding of complaints related to ETS in the workplace. Because it is readily identifiable, ETS is often <u>initially</u> blamed for IAQ problems (yet after investigation, reported exposures to ETS are directly associated with complaints in only two to five percent (2-5%) of sick-buildings).[1-4] If individuals are "stressed" by their work environment (influenced by such diverse factors as temperature, humidity, air movement, ergonomics, workload, personal problems, etc.), the mere visibility of ETS may provide a cue for a

complaint. Recent research by Winneke and colleagues indicates that an individual with a dislike of ETS will more readily, under actual exposure conditions, respond with annoyance symptoms.[17] (Ex. 3-1074) Researchers from the Illinois Institute of Technology also addressed the issue of nonsmoker perception of annoyance and irritation from ETS exposures. The study, involving over 250 subjects, reported that visual contact with a smoker increased the magnitude of adverse response to ETS among selected nonsmokers. The authors suggest that their conclusion "provides an inexpensive strategy of reducing complaints associated with ETS: eliminate visual contact between smokers and nonsmokers." (Ex. 3-31)

Precise quantitation of psychological variables and perceptions of comfort within the context of complaints about IAQ is extremely difficult. Nevertheless, the influence of those environmental variables upon IAQ complaints must be recognized and considered in a comprehensive approach to IAQ.

OSHA's Proposed Rule does not establish a significant risk of material impairment from IAQ problems in the nonindustrial workplace; data are available to establish significant risk from SBS and BRI in the workplace, but the Proposed Rule does not reference or document representative examples

Despite a number of submissions to the OSHA RFI docket on IAQ that document specific examples and studies on sick-building syndrome (SBS) and building-related illness (BRI) (e.g., Exs. 3-

500, 3-933, 3-955, 3-1053, 3-1054, 3-1073, 3-1074, 3-1183, 3-1185), the Proposed Rule provides only a cursory examination of the available data. (59 FR 15970-73). Specific case reports document hundreds of instances of BRI and SBS that often result in occupant deaths or debilitating illnesses. A sample of those buildings is summarized in Table I. The 287 specific cases of SBS/BRI were not in any way associated with ETS; in many of the buildings, smoking was prohibited altogether.

Studies and reports of SBS and BRI document thousands of cases of specific diseases and illnesses, yet OSHA only acknowledges the possibility of such instances and chooses, instead, to provide a theoretical, quantitative model that estimates the risk of headaches and upper respiratory symptoms from poor IAQ. (59 FR 15997) The model does not include instances of death and serious illness due to poor IAQ. OSHA's model projects potential cases of dry eyes, stuffy nose and headache, but it does not satisfactorily demonstrate a significant risk of material impairment from IAQ. Specific instances of mortality and morbidity due to poor IAQ in the nonindustrial workplace, on the other hand, are available (and were made available to OSHA in submissions to the RFI docket on IAQ). OSHA has not presented the "best available evidence" to support its own position on IAQ.

BUILDINGS THAT ARE REPORTEDLY SICK

No.	State	City	Diagnosis	Date	Building	Comment
1.	AL	Enterprise	SBS	1991	Coffee County Department of Human Resources	Allergic responses from nearly all 37 employees; building still causing health problems
2.	AK	Anchorage	SBS	1990	State Office Building (Henley v. Blomfeld Co.)	Carpet/HVAC; case settled
3.	AK		BRI	1992	Indoor skating rink	Freon leak and inadequate ventilation; one death, 33 injuries
4.	AZ	Globe	SBS	1993	Gila County Sheriff's Building	15 people hospitalized; suspect air-conditioning system drawing in bacteria from old carpeting
5.	AZ	Phoenix	SBS	1993	Corporation Commission	
6.	AZ	Phoenix	SBS	1990	Department of Economic Security	Unusual odor; 10-15 people experience burning eyes, sore throats, rashes, lethargy; chemical used in microfilm duplication machine is suspect; plan to clean air conditioning duct work
7.	AZ	Tucson	SBS	1988	Amelia Maldonado Elementary School	Sewer gases; carbon dioxide, chlorine, pollens, spores, other allergens; school eventually closed
8.	CA	Anaheim	SBS	1990	Pacific Volt	
9.	CA	Anaheim	Legion	1992	Western Medical Center	Emergency room staff members have disease; testing underway
10.	CA	Beverly Hills	SBS	1992	The Beverly Plaza Hotel	Formaldehyde; other chemicals
11.	CA	Burbank	SBS	1989	Lockheed Corp.	440 OSHA violations; 88 suing
12.	CA	Chino	SBS	1990	California Institution for Men	Workers Comp Claims; 50% of employees affected; cause unknown
13.	CA	Duarte	Legion	1986	City of Hope Medical Center	Cooling System
14.	CA	El Segundo	SBS	1985	El Segundo Airport Towers, Building C (Call v. Prudential)	Renovation; poor ventilation; case settled during trial
15.	CA	El Segundo	SBS	1991	Phoenix Computer Corporation	General allergic reactions from new carpets, furniture and paint; HVAC system leaked; 40 employees settled
16.	CA	Goleta	SBS	1988	Raytheon Co. (Buckley)	Synthetics, carpet glue, plastic partitions; settled for $625,000
17.	CA	Hayward	BRI	1982	Helen Turner Child Care Center	Chemicals from new roof; plaintiffs won class action suit against school, contractor and chemical manufacturer

18.	CA	Jackson	SBS	1991	Amador County Courthouse, District Attorneys Office	Possible ventilation problem; building newly renovated
19.	CA	Los Angeles	Legion	1988	UCLA Medical Center	8 infected; hot water system
20.	CA	Los Angeles	Legion	1988	Westwood Horizons residential facility	4 deaths; air conditioning unit
21.	CA	Los Angeles	SBS	1992	Pierce College library	Dirty ventilation ducts; HVAC hadn't been cleaned for 35 years; found dead animals, among other things, in ducts
22.	CA	Oakland	SBS	1991	Merritt-Peralta Medical Center	Test results pending; employees report hair loss
23.	CA	Oakland	BRI	1991	Alameda County Court House	Earthquake repairs; chemical fumes
24.	CA	Poway	SBS	1988	Midland Elementary School	Mold; general unsanitary conditions; students with allergies; tests inconclusive
25.	CA	Richmond	Legion	1990	Social Security Administration	Basement sink, 2 of 5 cooling towers; 3 lawsuits filed
26.	CA	Richmond	Legion	1991	Richmond Health Center	Test results pending
27.	CA	Riverside	SBS	1992	Riverside County Courthouse	Recently renovated; 15 ill
28.	CA	Sacramento	SBS	1982	Bateson Building (State Office Building)	Off-gassing from carpets and furnishings; building opened in 1981
29.	CA	Sacramento	SBS, Legion	1991	Twin Towers -- State Office Buildings CA Depts. of Health Services & Social Services	Leaks in buildings; dirty vents; poor ventilation; employee testified before Congress on IAQ; still being reported in 1993 respiratory problems and chronic fatigue 17 cases confirmed; CDC was asked to investigate; changes to ventilation system Study completed by CDC in 1994; over 600 reported fatigue; recommendations will come later
30.	CA	San Diego	SBS	1990	San Diego County Courthouse	Deaths and mysterious illness; asbestos cleared as cause
31.	CA	San Diego	SBS	1990	(Henchey v. Income Property Group Office Park Building, et al.)	Ventilation; suit settled for $65,000
32.	CA	San Diego	SBS	1984	Palomar College Library	Complaints of headaches, respiratory problems and concentration problems; plans to remove and replace current heating and ventilation system
33.	CA	San Francisco	Legion	1992	University of California Medical Center	Water contaminated; one death
34.	CA	San Francisco	BRI	1992	Langley Porter Psychiatric Institute (University of California at San Francisco)	Poorly designed ventilation system drawing fumes from parking lot

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35.	CA	San Francisco	SBS	1988	Bancroft-Whitney Co.	Over 900 employee complaints
36.	CA	San Francisco	SBS	1993	University-San Francisco Mission Center Building	General malaise, eye, nose and throat irritation; diisocyanate from an adjacent automobile body and paint shop; replace main air filtration system; cost for ventilation improvement - $500,000
37.	CA	San Pedro	SBS	1992	Banning High School	Fumes from nearby refinery entering school through ventilation system
38.	CA	Stanford	Legion	1983	Stanford University Medical Center	7 infected; incidents occurred separately following heart surgery
39.	CA	Sunnyvale	Legion	1990	Lockheed Missile and Space Co.	Two buildings closed
40.	CA	Westminster	TB	1993	LaQuinta High School	Outbreak of drug-resistant T.B. 292 students tested positive in 1993; 84 additional cases in 1994; CDC said faulty ventilation contributed to outbreak; HVAC has been repaired
41.	CA	Westwood	Legion	1977	Wadsworth Veterans Administration Medical Center	201 cases reported between 1977 and 1981; bacteria traced to drinking water supply
42.	CA		SBS	1983	Miller v. Lakeside Village Condominium Association	Tenant experienced allergies from mold infested apartment; case dismissed based on statute of limitations
43.	CO		SBS	1992	District Attorneys Office, 16 Jud. Dist. (Avery-Jorganson v. District Attorney)	
44.	CO		SBS, Radon	1990	(Brafford v. Susquehanna Corp.)	Risk of future cancer
45.	CO	Castle Rock	SBS	1991	Douglas County School District Administrative Offices	19 employee complaints
46.	CO	Denver	Legion	1992	St. Anthony Hospital Central	3 dead, 2 others infected; traced to hot water tank
47.	CO	Denver	SBS	1989	Federal Building	Numerous illnesses; health survey conducted
48.	CO	Ft. Collins	SBS	1992	Colorado State University	115 employee complaints of symptoms; cause is under investigation
49.	CO	Lamar	Legion	1989	Best Western Cow Palace	18 ill, three dead
50.	CO	Rocky Flats	BRI	1991	Rocky Flats Nuclear Weapons Plant (U.S. Department of Energy)	Plutonium in ventilation system; building closed
51.	CO	Westminster	SBS	1990	City Pool	Ventilation system; lifeguards suffering from hypersensitivity pneumonitis; contractors are being sued; 82 workers' comp claims filed
52.	CT	Bristol	BRI	1984	Police - Court Complex	Mold in ducts; employees experiencing SBS

						symptoms; experts suggested cleaning and rebalancing ventilation system and monitoring cleaning materials
53.	CT	Hartford	SBS	1992	Bulkeley High School	Students exhibit classic sick building syndrome symptoms
54.	CT	Hartford	SBS	1991	One Myrtle Street (Padgett v. Capital West Associates, et al.) (Potential class action)	Bad ventilation, general uncleanliness, 40 plaintiffs reportedly asking for $1 million each
55.	CT	Hartford	SBS	1991	U.S. Post Office (Mendenhall, et al. v. Kerin; Clarkin v. Kerin) (potential class action)	Eye and lung problems
56.	CT	Middletown	legion	1993	Connecticut Valley Hospital	Source of infection still unknown; examination and cleaning of air-conditioning system as a precaution; attempt to determine whether 4 other cases of pneumonia are related to same problem
57.	CT	New Britain	SBS	1984	New Britain High School	Expert recommended upgrading ventilation system
58.	CT	New Haven	Legion	1986	Hospital of St. Raphael	Hot water pipes
59.	CT	New Haven	BRI	1985	Yale Medical School, Dept. of Epidemiology and Public Health	New carpet glue
60.	CT	Stamford	SBS, Legion	1990	West Main Street Shelter	Inadequate ventilation; moisture in walls; rat and bug infestation; building to be demolished
61.	CT	Stratford	Legion	1994	Stratford Group Home for the Retarded - Grasso Center	4 residents, 1 employee diagnosed with pneumonia; 1 of the 5 has been diagnosed with Legionnaire's disease; sources of water being checked
62.	CT		SBS	1991	Connecticut insurance company	Class action suit alleging that building conditions adversely affected its employees' health
63.	DC		SBS	1990	EEOC Headquarters	Fungus in ventilation; building to be gutted
64.	DC		SBS	1989	U.S. Dept. of Interior (Perkins v. Maitico Operating Co.)	Excessive heat, humidity and bacteria; case settled
65.	DC		BRI	1988	U.S. Information Agency	Lead in water
66.	DC		SBS	1986	General Services Administration Building	Mysterious ailment sent 20 to hospital
67.	DC		Graves	1991	White House	Lead pipes/paint
68.	DC		Legion	1991	SSA Payment Center	

69.	DC		SBS	1989	Parklawn Building, U.S. Dept. of HHS	
70.	DC		SBS, Legion	1989	Madison Building, Library of Congress	Tests ordered
71.	DC		SBS	1988	EPA Waterside Mall (Bahura v. SEW Investors)	New carpet; December 1993 bifurcated trial resulted in award of damages to 5 plaintiffs by jury; twelve plaintiffs still awaiting trial
72.	DC		SBS	1989	U.S. Housing and Urban Development Building	
73.	DC		SBS	1989	Frances Perkins Building, U.S. Labor Dept.	
74.	DC		BRI	1986	Hubert Humphrey Office Building	25% developed hypersensitivity pneumonitis; leaking cafeteria pipes
75.	DC		SBS	1992	National Museum of American History, Smithsonian Institution	Asbestos fibers floating in corridors from ventilation system
76.	DC		SBS	1993	U.S. Information Agency (USIA) Library	Air was rendered unbreathable; inspection of Colonial parking garage, below revealed fan venting carbon monoxide straight into library; negotiations about who will move the vent and pay for it, is underway
77.	DC		SBS	1988	National Oceanic and Atmospheric Administration Building, No. 1	Complaints of headache, flu-like symptoms; inadequate ventilation system; presence of phenol found; 4 employees received workers' compensation for illnesses; considering improving ventilation and removing phenol
78.	FL		SBS	1990	(Stillman v. South Florida Savings & Loan)	Breach of contract case
79.	FL		SBS	1991	(Eagle-Picher Industries v. Cox)	Florida District Court
80.	FL	Bartow	SBS	1991	Imperial Polk County Judicial Complex (Polk County v. Barton-Malow Co. & W. Wade Setliff)	300 have filed workers' comp claims; mold, high humidity, faulty air conditioning system; $18 million lawsuit filed against contractors; employees readying suit against County and others; new roof required; cost to fix building estimated at $22.5 million; ventilation system to be replaced
81.	FL	Brooksville	SBS	1992	Hernando County Courthouse	Inadequate ventilation, leaky roof, sagging ceilings, moldy walls and carpets; county to begin $2 million renovation; employees want to be moved out
82.	FL	Dade City	SBS	1992	Eastside County Courthouse	Need to improve HVAC maintenance
83.	FL	Deerfield Beach	SBS	1991	House of Insurance	Complaints filed

84.	FL	Deland	BRI	1983	Deland Courthouse	Judge experiences vertigo; is hospitalized; mold growths, wetness problems, poor ventilation; county seeking $250,000 to repair roof and air-conditioning system
85.	FL	Deland	SBS	1987	Volusia County Courthouse	Workers complain of respiratory tract problems; mold, mildew on walls, ceilings and in air duct system; many areas have poor ventilation, high carbon dioxide levels; plan to replace roof and air conditioning system By 1994 five circuit court employees intend to sue; whole building to be evaluated
86.	FL	Ft. Lauderdale	SBS	1988	Goldman v. Broward County	Workers' Compensation
87.	FL	Ft. Lauderdale	SBS	1991	South Florida Savings Bank (Zandman v. Atria, et al.) (Five additional cases filed)	Nine defendants; destroyed immune systems
88.	FL	Ft. Lauderdale	BRI	1991	Broward County School (Rosenfeld v. School Board of Broward County)	Paint fumes
89.	FL	Ft. Lauderdale	SBS	1991	Broward County Library	Complaints filed
90.	FL	Ft. Lauderdale	SBS	1991	Paragon Building	Complaints filed
91.	FL	Ft. Lauderdale	SBS	1991	Broward County Airport Control Center	Complaints filed
92.	FL	Fort Meade	SBS	1991	Fort Meade City Hall	Inadequate HVAC system; mold; mildew
93.	FL	Fort Pierce	SBS	1990	State Attorneys' Office Building	Improper HVAC caused mold/mildew build-up; employees have filed suit against St. Lucie County claiming permanent injuries
94.	FL	Gainesville	SBS	1986	Veterinary School, Univ. of Florida	Ventilation system; mold and organic chemical pollution
95.	FL	Gainesville	SBS	1992	Tacachale Community Building	Inadequate HVAC caused mold/mildew on walls and ceiling; building houses 50 mentally impaired patients
96.	FL	Gainesville	SBS	1992	Santa Fe Community College, Bldg. C	High humidity, mold, mildew
97.	FL	Gainesville	BRI	1992	Library West, Univ. of Florida	Chemical fumes; faulty ventilation
98.	FL	Hernando County	SBS	1992	J.D. Floyd Elementary School	Teachers and students have allergy-type symptoms; school was thoroughly cleaned and carpets replaced
99.	FL	Inverness	BRI	1993	Citrus Springs Elementary School	Students felt sick at school; respiratory complaints
100.	FL	Kissimmee	Legion	1992	The Hyatt - Orlando Hotel	Tests are pending; cases confirmed

101.	FL	Largo	Legion	1994	Pinellas County Criminal Courts Complex Jail	2 people tested positive for Legionnaire's disease; bacteria were isolated in an air conditioning unit; bacteria found in a cooling tower on roof of maximum security facility; evacuations may be necessary
102.	FL	Lauderdale Lakes	SBS	1991	Lauderdale Lakes City Hall	Complaints filed
103.	FL	Lauderhill	SBS	1991	Lauderhill Mall, State of Florida Offices	Complaints filed
104.	FL	New Port Richey	SBS	1992	Westside County Courthouse	Need to improve HVAC maintenance
105.	FL	Oakland Park	SBS	1991	Northridge Medical Plaza, V.A. Clinic (Clavon v. Northridge Medical Plaza)	Dangerous mold in air
106.	FL	Oldsmar	SBS	1992	Oldsmar Elementary School	Moldy, moist air; school was closed for repairs; in 1993 school reopened
107.	FL	Orlando	BRI	1992	West Orange High School	Amount of mold is 35 times higher than test results show; complaints include headaches, fatigue, sinus infections, coughing; due to inadequate and/or poorly operating air-conditioning
108.	FL	Orlando	BRI	1992	Cypress Creek High School	IAQ problems
109.	FL	Orlando	BRI	1992	Walker Middle School	IAQ problems
110.	FL	Orlando	BRI	1992	Liberty Middle School	IAQ problems
111.	FL	Orlando	BRI	1992	Chickasaw Elementary School	IAQ problems
112.	FL	Orlando	BRI	1992	Blankner Elementary School	IAQ problems
113.	FL	Orlando	BRI	1991	Palmetto Elementary School	Teacher diagnosed with chronic fungal sinusitis; air in buildings tainted by chemical vapors, molds, and poor ventilation
114.	FL	Orlando	SBS	1993	Hyatt-Regency International Airport Hotel	Mildew present in 159 of its 445 rooms; clean up is planned
115.	FL	Orlando	SBS	1993	Orange County Government Building	Almost a dozen workers have been affected; solution estimated at millions of dollars; 10 workers were awarded disability - 2 permanent; health officials say Florida is nation's "sick building" capital; ventilation system inadequate
116.	FL	Palm Bay	SBS	1993	Discovery Elementary School	"Flu-like" symptoms; clean up of air conditioning system; burning eyes, upper respiratory problems, "onion-type" odor may be due to sulfate
117.	FL	Palm Harbor	SBS	1992	Curlew Creek Elementary School	Mold and mildew from high humidity levels; IAQ firm recommended new carpet, upgrading

						HVAC and more frequent cleaning; six teachers have received workers' compensation due to respiratory problems from poor IAQ
118.	FL	Pensacola	SBS	1990	Escambia County Dept. of Public Health	Mold, mildew and poor ventilation
119.	FL	Port St. Lucie	SBS	1989	Bayshore Elementary School	Inadequate HVAC; students and teachers symptomatic
120.	FL	St. Petersburg	SBS	1992	J.D. Floyd Elementary School	Carbon dioxide levels exceeded industry standards; bacteria and humidity at unacceptable levels; air-conditioning thermostats malfunctioned
121.	FL	St. Petersburg	SBS	1993	River Ridge School	Teachers complain of runny noses, nasal congestion, and odors; 2 buildings in question; ventilation system targeted
122.	FL	St. Petersburg	SBS	1993	Bloomingdale High school	Students and staff complained of persistent odor; reports of headaches, nausea and allergies; spending $2 million for dehumidifying, cleaning air conditioning ducts
123.	FL	St. Petersburg	SBS	1993	Gaither High School	Attempts to improve air quality due to complaints by students and staff
124.	FL	St. Petersburg	SBS	1993	Pinellas County School	Closed school after investigating complaints; determined that air quality posed a health problem
125.	FL	Spring Hill	SBS	1992	Pine Grove Elementary School	School was scrubbed, caulked, carpet replaced and drainage system changed
126.	FL	Stuart	SBS	1989	Martin County Courthouse Complex	Mold, algae, fungus in air conditioning ducts; 10 treated at hospital; toxic fungi discovered in late 1992; building closed; dozens being tested for health problems; "lawsuits are flying"
						By 1993, 200 people complained; contaminated air conditioning system; leaks in roof; 21 employees filed claims; building closed
						In 1994, construction underway to replace building's exterior envelope, roof, all interior surfaces and all air handling units
127.	FL	Tallahassee	SBS	1992	Doyle Carlton Building	Chemical fumes from building clean-up
128.	FL	Tampa	SBS	1992	Hillsborough County Crisis Center	Dirty ventilation ducts; closed fresh-air intakes; high carbon monoxide levels

129.	FL	Tampa	SBS	1992	University Community Hospital	OSHA investigated following 20 IAQ complaints; cause undetermined
130.	FL	Tampa	SBS	1989	Office of Disability Determination	Pigeon skeletons and bird droppings found in ventilation system
131.	FL	Tampa	SBS	1992	George Edgecomb County Building	Violation of air quality standards; cited by state
132.	FL	Tampa	Legion	1993	Lockhart Elementary School	Suspected legionella; investigations are on-going; pigeons may be a source of problem In 1994, consultants are still monitoring school
133.	FL	University Station	SBS	1991	John and Grace Allen Administration Building University of South Florida	Inadequate ventilation; water damaged carpets and ceiling tile; 77 have filed workers' comp claims; NIOSH investigated
134.	FL	West Palm Beach	SBS	1991	City Hall	Undersized fresh air intakes
135.	FL	West Palm Beach	SBS	1991	Palm Beach County Governmental Center	Complaints filed
136.	FL	Ybor City	SBS	1991	Lozano Building Hillsborough County Environmental Protection Commission	High carbon dioxide levels, inadequate ventilation; termite infestation creating a dust problem
137.	GA		SBS	1990	Administration Building, University of Georgia	NIOSH investigated, found mold, low humidity and inadequate ventilation; building is smoke-free
138.	GA	Atlanta	SBS	1980	101 Marietta Tower Bldg.	Dust and humidity; NIOSH investigated
139.	ID		SBS	1988	North Idaho College, Hedland Vocational Building	Fumes entering ventilation system and circulating throughout building from shop classroom
140.	ID	Boise	BRI	1994	Idaho Ice Arena	Five hockey players hospitalized -- from inhaling poisonous fumes from ice-shaving machine; 22 others treated then released; rink closed to check ventilation system; lawsuits pending
141.	IL	Chicago	SBS	1991	Wrigley Building	Ventilation system; exhaust from boats docked below building
142.	IL	Chicago	Legion	1991	Social Security Administration Bldg.	Bacteria found; evaluated after outbreak in Richmond, CA
143.	IL	Chicago	SBS	1992	Cook County Vital Statistics Bureau	Poor ventilation, paper mites in basement; workers complaining of rashes, respiratory problems

144.	IL	Chicago	SBS	1993	Chicago's Daley Center	Cancer-causing chemicals found in soot spewing from air vents; complaints of asthma, bronchitis, dizziness, nausea and fatigue; problem result of a faulty boiler; benzopyrene and benzanthracene identified
145.	IL	Chicago	BRI	1993	Hinsdale Hospital (Elmwood Hall)	Hospital employees complain of burning in sinuses and ears; emergency room care required; faulty ventilation
146.	IL	Chicago	SBS	1993	St. Charles Community High School	Complaints of nausea, headaches and flu-like symptoms; ventilation problems
147.	IL	Chicago	Legion	1993	University of Illinois Hospital	Bacteria in facility's water system; transplant patient dies of Legionnaire's Disease
148.	IL	Chicago	SBS/Legion	1993	University of Illinois at Chicago (Education, Communications and Social Work Building)	Ventilation may be faulty; professor died of pneumonia; more than a dozen employees complained of feeling ill
149.	IL	Chicago	BRI	1994	Bolingbrook's Addams Middle School	Tests by industrial hygienists indicated hydrogen sulfide emitted from rooftop vents led to over 2 dozen students being hospitalized; ventilation system needs re-routing
150.	IL	Chicago	SBS	1982	Rolling Meadows Junior High School	5 cancer-related deaths of staff since 1982; medical experts hired to determine possible link to Plumgrove school deaths
151.	IL	DuPage	SBS	1993	GlenEllyn Elementary School	Formaldehyde and carbon dioxide detected in a portable unit; deficiency in ventilation system
152.	IL	St. Charles	SBS	1992	St. Charles Community High School	Grievance filed with teachers' union; IAQ tests showed high carbon dioxide levels, inadequate fresh air, high dust levels
153.	IL	Hanover Park	SBS	1993	Johnson School	Complaints from teachers and pupils; IAQ company to test for carbon dioxide, humidity levels
154.	IL	Hinsdale	SBS	1992	Hinsdale Middle School	Students experiencing illness; IAQ firm has been hired to evaluate; in 1993 recommend redirecting exhaust air ducts to discharge above roof be done
155.	IL	Hinsdale	SBS	1992	Lane Elementary School	Carpet emissions were blamed for illnesses; problems allegedly solved by cleaning carpets

156.	IL	Palatine	SBS	1982	Plumgrove Junior High School	5 staff dead between 1982-1992; several developed breast cancer and some respiratory ailments during 1993-94; asbestos floor tiling and pipe insulation removed in 1993
157.	IL	Waukegan	BRI	1992	Lake County Health Department (Belvedere Medical Building)	Nurses report sinus problems and occasional shortness of breath; dust and mold in the ventilation system; cleaning of carpets and regular inspection of ventilation system will occur
158.	IL	Wheaton	SBS	1992	DuPage County Courthouse (County of DuPage v. NOK et al.) (Bostick v. County of DuPage)	Tests inconclusive; building newly constructed; 20 employees hospitalized; courthouse closed for repair; scheduled to reopen March 1993; county filed suit against architect and builders seeking $5.5 million
159.	IN	Evansville	Legion	1985	St. Mary's Medical Center	
160.	IN	Indianapolis	Legion	1988	Methodist Hospital	10 cases, 5 deaths; water system
161.	IN	Kokomo	SBS	1990	Western Primary Elementary School	76% of students exhibited symptoms and transferred to another building; HVAC was replaced in mid-1992
162.	IN	Lebanon	Legion	1993	Harney Elementary School	Suspected Legionnaire's disease - related pneumonia of 17 students
163.	IN	Russiaville	SBS	1991	West Elementary School	HVAC; NIOSH investigated
164.	IN		BRI	1992	Indiana office complex	Change in insecticide used at adjoining warehouse, dirty ventilation system and inadequate air intakes; building was decontaminated
165.	IA	Iowa City	Legion	1981	University of Iowa Hospitals	"Water bug"; 24 cases
166.	IA	Ottumwa	SBS	1990	Wapello County and Iowa Depts. of Human Services Building (Bloomquist v. Wapello County)	Pesticides in carpet, HVAC inadequate, leaking sewer line
167.	KS	Belleville	BRI	1990	Agricultural Stabilization and Conservation Services; Soil Conservation Service (USDA Agencies) (Dreeson v. WW Henry Co.)	Carpet glue; cases dismissed
168.	KS	Wichita	SBS	1989	Pioneer TeleTechnologies	62 ill; tests inconclusive
169.	KY	Bowling Green	SBS	1988	Richardsville Elementary School	Inadequate ventilation and hog lot next door; high miscarriage rate

170.	KY	Frankfort	BRI	1993	Cabinet for Human Resources	Test revealed microbes that can cause strep throat, staph. infections and sinus problems; elevated carbon dioxide; dry air; upgrade ventilation system
171.	LA	Baton Rouge	SBS	1992	Valley Park Administrative Center, East Baton Rouge Parish	Dirt, dust, mold and fungi in air ducts; building sits atop a landfill
172.	LA	Baton Rouge	SBS	1991	Pleasant Hall, Louisiana State Univ.	Ventilation system; black particles falling from vents; mold
173.	LA	Bogalusa	Legion	1988	Winn-Dixie Grocery Store (Crowe v. Winn-Dixie)	Vegetable mister; 33 contracted disease
174.	LA	New Orleans	BRI	1993	New Orleans' City Hall	Mold, mildew, carbon monoxide, soggy ceiling panels (shedding asbestos); multiple employee complaints; air conditioning system malfunctions; mechanical system in basement the worst; raw sewage, roaches, drums containing chemicals; needed a comprehensive building management program
175.	ME		SBS	1991	Maine Department of Transportation	Building being monitored, test results pending
176.	MD	Baltimore	SBS	1989	Lighting limited	New carpet blamed
177.	MD	Baltimore	Legion	1993	Sarah M. Roach Elementary School	1 teacher, 12 students developed chronic illness; ventilation and heating problems
178.	MD	Baltimore	SBS	1978	General Hospital, Carroll County	Poor IAQ; ventilation system needs to be balanced; spraying with biocide was no help; roof needs repair to reduce mold
179.	MD	Bethesda	Legion	1984	Bethesda Naval Hospital	Showerheads and faucets
180.	MD	College Park	Legion	1986	University of Maryland	Bacteria in utility tunnel and air conditioning system; bacteria found in 3 buildings
181.	MD	Hagerstown	Legion	1990	Western Maryland Center State Hospital	Hot water system; 6 ill
182.	MD	Rockville	Legion	1991	U.S. Public Health Service Building	Test results pending
183.	MD	Waldorf	SBS	1987	Thomas Stone High School	Blackish mold
184.	MD	Woodlawn	SBS	1991	U.S. Health Care Financing Administration, Meadows East Building	Fungus contamination suspected; earlier incident in 1986
185.	MD	Woodlawn	SBS	1986	City Office Building	Complaints followed asbestos removal
186.	MA	Andover	SBS	1991	West Elementary School	Mold in basement; ventilation; in 1993 reports mold problem has been rectified

187.	MA	Boston	Legion	1988	Metropolitan State Hospital	
188.	MA	Boston	SBS	1993	Rowes Wharf Office Highrise	Law firm tenant says insulation fibers are coming through buildings ventilation system; building managers say law firm's carpet is emitting VOCs; firm moving out, managers vow to sue
189.	MA	Boston	SBS/BRI	1993	University of Massachusetts, Phyllis Wheatley Building	Students nauseous and dizzy; fumes entered through ventilation system In 1994, 27 people sought medical treatment; unexplained nausea, burning lips and tight throats; 5 employees, 2 students treated for airborne illness; (to date 50 employees have been affected); $550,000 for renovation of ventilation system
190.	MA	Boston	SBS/BRI	1993	University of Massachusetts, Healey Library	IAQ problems
191.	MA	Boston	SBS	1993	College of Public and Community Service	Secretary hospitalized after experiencing severe symptoms; inhalation of chemical irritant through an air duct; improper installation of ventilation, heating and air conditioning systems
192.	MA	Boston	SBS	1993	Boston College, Merkert Chemistry Center and Silvio Conte Forum	Chemicals vented through structure; buildings evacuated pending further investigations
193.	MA	Brigham	SBS	1994	Women's Hospital	47 nurses on disability; 300 of 1800 nurses reported health problems; one dietitian, with asthma, died; OSHA investigating
194.	MA	Cambridge	SBS	1982	Cambridge Rindge & Latin High School Arts Building	HVAC; elevated carbon dioxide levels; birds nesting in air ducts; moldy carpet; teacher testified in Congress in 1988; litigation pending against contractors
195.	MA	Cambridge	SBS	1980	Cambridge High and Latin School	Building was torn down and merged with Cambridge Rindge and Latin School
196.	MA	Cambridge	SBS	1991	Tobin School	School built over former dump site; inadequate ventilation; elevated methane levels
197.	MA	Fall River	Legion	1993	Notre Dame Church	Source could be funeral parlor adjacent to church; 3 cases confirmed
198.	MA	Fall River	Legion	1994	Stafford Apartment Complex	Legionella bacteria found in cooling tower; 11 people infected

199.	MA	Danvers	SBS	1993	Dunn Middle School	Lethargy, headaches; inadequate ventilation
200.	MA	Danvers	SBS	1993	Smith Elementary School	Lethargy, headaches; poor ventilation
201.	MA	Lynn	SBS	1993	Lynn City Hall	Employees request IAQ inspection
202.	MA	Norwood	SBS	1990	Polaroid	Leaking generator exhaust fumes, insufficient fresh air
203.	MA	Roxbury	SBS	1994	T-Cell Sciences, Inc.	45 out 126 employees affected by skin rashes and respiratory irritations; suspect ventilation and maintenance issues; some employees were briefly relocated
204.	MA	Roxbury	SBS	1994	Registry of Motor Vehicles	Twenty-four employees complained of nausea, skin irritation, and headaches; inadequate ventilation near a large printer; ceiling debris and heating pumps also blamed; registry officials "vowed action"
205.	MA	Orleans	BRI	1994	Charles Moore Arena	52 students at a prep school figure skating camp were overcome by fumes from ice resurfacing machine; in summer, "some rink managers shut down ventilation systems to keep hot air out."
206.	MI	Detroit	SBS	1986	Madison Center (Court Building)	New renovations; 100 hospitalized
207.	MI	Detroit	Legion	1985	Airport Hilton Inn	Bacteria in air conditioning
208.	MI	Jackson	Legion	1993	Southern Michigan Prison	31 inmates stricken; 3 died; spread through ventilation system
209.	MI	Pontiac	Legion, SBS	1991	Oakland County Complex	Investigation underway, test results are pending; also upper respiratory infections reported and problems with ventilation system
210.	MN	Brainerd	SBS	1991	Brainerd Community College (Room 19)	Teachers complain of nausea, headaches, dizziness, foul odors; ventilation system inadequate; air-distribution problem
211.	MN	Duluth	SBS	1993	University of Minnesota, Library	12 employees, 1 student; complaints of respiratory illness; dust and mold spores; very poor ventilation that could have bred mold; contractor hired to clean
212.	MN	Minneapolis	SBS	1993	Minnesota Health Department	Fresh air intake directly above the building's loading dock where garbage trucks run their motors to compact trash

213.	MN	St. Paul	SBS	1993	Northern Service Center Building	Complaints of eye irritation, nasal congestion neck and back pain, may be cancer-related; 17 out of 47 employees reported diagnosed cancer; private consultant to analyze IAQ
214.	MN	St. Paul	SBS	1993	Centennial Building	Fumes from trucks idling at the loading dock drawn into building
215.	MN	St. Paul	BRI	1994	St. Paul Hockey Arena	116 cases of respiratory illnesses out of 167 players, cheerleaders, and band members in attendance
216.	MO	Centralia	BRI	1986	(Pinkerton v. Temple Industries Inc.)	Indoor formaldehyde exposure; $ 16.2 million jury award
217.	MO	Dixon	BRI	1993	Dixon Elementary School	Unexplained rashes which go away after leaving school reported by teachers and students; tests still underway; high dust levels, low humidity, inadequate ventilation, dead birds above ceiling; problems disappeared following thorough cleaning and airing out
218.	MO	Jefferson City	SBS	1986	Truman State Office Building	Several dozen employees sick
219.	MT	E. Helena	SBS	1981	Tri-Valley Credit Union Building	Tests inconclusive
220.	NE	Lincoln	Legion	1986	Federal Building	
221.	NV	Carson City	SBS	1987	State Capitol Building	Tests inconclusive; legislators have allergies; could not find cause
222.	NV	Lake Tahoe	BRI	1991	Harvey's Wagon Wheel (Scott v. Harvey's Wagon Wheel, Inc.)	Pesticides; 15 have lingering illness
223.	NH	Concord	SBS	1991	Beaverbrook Elementary School	Poor indoor air quality; new HVAC system installed
224.	NH	Nashua	SBS	1987	New Searles Elementary School	Damp air, molds, ventilation
225.	NJ	Atlantic City	BRI	1991	Bally's Park Place Hotel and Casino (Secretary of Labor v. Bally's)	Iodine emissions from cold-water glass washing machine
226.	NJ	Camden	SBS	1990	Camden County Administration Building (Lipsitz v. Scallop Thermal Management)	
227.	NJ	Flemington	Legion	1989	Hunterdon Central Regional High School	
228.	NJ	Jersey City	SBS	1992	590 Newark Ave. Bldg	Faulty HVAC system; water leaks; fumes from repair work

229.	NJ	Mahwah	SBS	1991	Municipal Offices	High carbon dioxide levels; inadequate ventilation
230.	NJ	Mahwah	SBS	1991	Ramapo College	Ventilation system; contaminated laboratory air
231.	NJ	Red Bank	SBS	1987	Bell Communications Research Building	Hydraulic elevator system; poor general ventilation; high levels of VOCs found
232.	NM	Albuquerque	Legion	1991	Social Security Administration	Bacteria found; evaluated after outbreak in Richmond, CA
233.	NY	Albany	SBS, Legion	1989	West Mall Office Plaza NYS Dept. of Estate & Gift Tax (Workers' compensation claim)	Employee claim; other employees ill; mold in ventilation system
234.	NY	Albany	SBS	1990	Building 8, W.A. Harriman State Office Campus Department of Taxation and Finance	Heating units were leaking fluid; other test results pending; building was scrubbed; building sick again in late 1991; union wants building closed In 1993 heating, air conditioning and ventilation systems under repair; invasion of insects; poorly ventilated; inadequately cleaned
235.	NY	Albany	SBS	1990	State Legislative Office Building (LOB)	LOB has been dubbed the "L.O.B.-gionnaire's disease"; workers complain about poor IAQ and say they are symptomatic; Health Department inspected but office of General Services did not adopt any recommendations for improvement; legislators plan to introduce IAQ legislation in response
236.	NY	Brooklyn	Legion	1984	Downstate Medical Center	Air conditioning tower
237.	NY	Buffalo	BRI	1983	Roswell Park Cancer Institute	10 patients, all of whom died, contracted infection from aspergillus bacteria; 4 families won judgments after the deaths were linked to a faulty ventilation system; the hospital had another outbreak of aspergillus infection in October 1992 which is claimed to not be linked to the HVAC system In 1992 rooftop exhaust from labs discharged chemical compounds that were heavier than the air and infiltrated ventilation system
238.	NY	Buffalo	BRI	1993	West Seneca Educational Center Print Shop	Complaints of rashes, nosebleeds, headaches; recommended lower vent hood, increase chimney height, to improve ventilation; shop closed in January; reopened in March

239.	NY	Cheektowaga	Legion	1990	St. Joseph Hospital	Five contracted disease; 3 dead; bacteria found in hot water system
240.	NY	Delmar	SBS	1992	Department of Taxation and Finance	407 people diagnosed with arthritis or asthma; 43 former state employees have sued; class-action suit
241.	NY	Great Neck	SBS	1992	11 Grace Avenue Office Building	High concentrations of dirt particles; low humidity
242.	NY	Hauppauge	SBS	1991	State Office Building	Lack of fresh air; clogged ventilation ducts
243.	NY	Helmuth	Legion	1993	Collins Correctional Facility	2 inmates have contracted Legionnaire's disease
244.	NY	Hudson County	BRI	1989	Whitney Young Elementary School	Chromium residue
245.	NY	Long Island City	SBS	1992	Community Alternatives System Agency	Dirty ventilation system; several workers have filed union grievances
246.	NY	Nassau	SBS	1992	Nassau Community College	Teacher testified at state legislative hearing on IAQ
247.	NY	NYC	SBS	1988	100 Gold Street Building NYC Department of Housing Preservation and Development	Inadequate ventilation; renovation
248.	NY	NYC	Legion	1987	General Services Administration	Water tower was decontaminated
249.	NY	NYC	Legion	1985	New York Times Company	Six legionnaires; 29 respiratory illness
250.	NY	NYC	SBS	1991	Brandeis High School	Inadequate ventilation; no air conditioning or humidifying system and no windows
251.	NY	NYC	SBS	1992	Jewelry Store	Mold in ventilation system
252.	NY	NYC	SBS	1992	Columbia University, Career Services Center	Carbon monoxide from trucks idling near fresh air intakes; hot and cold spots from poorly operating temp control system; levels of black particles from dirty ventilation system; bug infestation
253.	NY	NYC	SBS	1993	Fiorello LaGuardia High School for the Performing Arts	Teachers and students are symptomatic; United Federation of Teachers has asked the Board to study problem
254.	NY	NYC	SBS	1993	Norman Thomas High School	Teachers and students are symptomatic; United Federation of Teachers has asked the Board to study problem

255.	NY	NYC	Legion	1994	Celebrity Cruise Ship	6 passengers contracted Legionnaire's disease; one man died, 2 in critical condition; 24 suspected cases; source appears to be ship's water system; a class-action suit seeking $150 million has been filed in Federal District Court in Manhattan
256.	NY	Queens	SBS	1992	Emergency Services Building	Fiberglass particles in air; insects; rodent droppings; general uncleanliness
257.	NY	Rochester	SBS	1984	Eastman Kodak	Fungus in ventilation system; 115 sick
258.	NY	Rochester	Legion	1990	Rochester General Hospital	Hot water system
259.	NY	Ronkonkoma	SBS, Legion	1992	County Building	Inadequate ventilation; lawsuit filed
260.	NY	Smithtown	SBS	1992	Smithtown Public Library	Inadequate ventilation
261.	NY	Wappingers Falls	SBS	1983	Wappingers Falls High School	Inadequate ventilation
262.	NY	Wappingers Falls	SBS	1983	Van Wyck Junior High School	Inadequate ventilation
263.	NY	Westbury	SBS	1992	New York Terminal Radar Approach Control	High heat, generated by electronic equipment; poor ventilation; general uncleanliness; center is busiest air traffic control in the U.S.
264.	ND	Fargo	SBS	1983	Southeast Human Service Center (Eiseman v. Southeast Human Service Center)	SBS/MCS lawsuit; employee reinstated with back pay; IAQ testing by state showed IAQ within OSHA standards
265.	OH	Cincinnati	SBS	1991	Alms & Doepke Building (Beck v. A&D Limited Partnership)	Defective sewers, dirty ventilation, inadequate fresh air; NIOSH did site survey
266.	OH	Cincinnati	SBS	1991	Goodall Building (Martinez v. Goodall Properties, Ltd.)	HVAC systems
267.	OH	Cincinnati	SBS	1981	(Beebe v. Burlington Industries, Inc.)	New carpet
268.	OH	Columbus	SBS	1990	Northland Terrace Nursing and Rehabilitation Center	Inadequate ventilation in laundry facility; perfumed diapers; NIOSH investigated
269.	OH	Dayton	SBS	1990	Wright Patterson Air Force Base	Lack of fresh air and carbon dioxide build-up
270.	OH	Dayton	Legion	1992	VA Medical Center	Bacteria located in water supply; 2 dead, 1 being treated

271.	OH	Toledo	BRI	1992	Family Court Center	14 people treated by emergency medical personnel; burning eyes, breathing problems; 100 people evacuated; OSHA inspected; need to raise main air intake which was bringing exhaust fumes/other contaminants into building; in 1993 officials said continuous air monitoring will take place
272.	OK	Muskogee	SBS	1990	CH Haskell Building, Oklahoma State University	Cooling tower; University to replace it
273.	OR	Portland	SBS	1992	Multnomah County Emergency Communications Center	Office is in former bomb shelter atop Kelly Butte; tests inconclusive; inadequate ventilation suspected; employees ill
274.	OR	Salem	SBS	1984	State Commerce Building	Tests inconclusive; suspected are transient fumes and "psychogenic factor"; over 60% of employees exhibited symptoms
275.	PA	Colwyn	BRI	1992	Colwyn Elementary School	Fungus-infested classrooms; students moved to new location; school reopened in 1993
276.	PA	Greensburg	SBS	1992	Westmoreland Manor County Home for the Elderly	Tests pending; employees ill
277.	PA	Harrisburg	BRI	1991	Sporting Hill Elementary School	Solvent used during asbestos removal
278.	PA	Kulpsville	Legion	1992	Penn Fishing Tackle Mfg. Co.	Manufacturing process blamed; 3 have contracted disease
279.	PA	Philadelphia	Legion	1991	Social Security Administration	Bacteria found; tested after outbreak in Richmond, CA
280.	PA	Philadelphia	SBS	1993	M. Thomas Stone Library Bryn Mawr College	Three say they have MCS following renovation; lawsuits have been filed against contractors
281.	PA	Pittsburgh	Legion	1992	Presbyterian University Hospital	4 deaths; seven confirmed cases; bacteria traced to water supply
282.	PA	Pittsburgh	Legion	1993	Gateway View Plaza	7 people contracted disease; 1 died; ventilation system needed adjustment
283.	PA	West Chester	SBS	1992	West Chester University	Employees surveyed; 20% are symptomatic; poor ventilation blamed
284.	PR	Hato Rey	SBS	1990	HUD Office	Stagnant water in air-handling units;
285.	RI	Providence	Legion	1983	Rhode Island Hospital	Bacteria in exposed water in cooling tower came in through open windows
286.	RI	Providence	SBS	1992	Green State Airport Terminal	Dirty air ducts; inadequate ventilation; airplane exhaust reaching air intake vents
287.	RI	Providence	Legion	1993	Veteran's Administration Hospital	One patient died of Legionnaires disease; CDC sent to investigate

288.	TN	Nashville	SBS	1991	Tennessee State Museum	Leaky walls; carpets sprouted mushrooms
289.	TN	Nashville	SBS	1991	(Metro) Courthouse	Filters and heating and cooling vents; judge suffering from allergies
290.	TN	Nashville	SBS	1991	Nashville Federal Building	Unidentified fumes; offices were evacuated
291.	TN	Nashville	SBS	1990	Arista Records Nashville Headquarters	14 employees with symptoms; company moved out of building
292.	TX	Austin	SBS	1991	Texas Dept. of Housing and Community Affairs	Partition furniture, carpet, lack of fresh air, sewer gases; Texas trying to rectify to insulate from liability
293.	TX	Austin	SBS	1990	Austin Hospital (Hughes v. City of Austin)	Ventilation problems; airborne chemicals; unidentified dust; defense verdict
294.	TX	Austin	SBS		Texas Board of Pardons and Paroles	Mold, contaminated carpets, leaking sewer gases, ozone pollution from copiers
295.	TX	Baytown	SBS	1992	Lamar Elementary School	Infestation of mold and mildew in air conditioning systems; affected area dubbed "death wing"
296.	TX	Baytown	SBS	1992	Ashbel Smith Elementary School	Tests found fungus; six classrooms were relocated; building to be closed
297.	TX	Baytown	SBS	1992	Bowie Elementary School	Tests are pending
298.	TX	Dallas	BRI	1992	Ferrell Center, Baylor Univ.	Arena evacuated; carbon monoxide fumes from gusty wind and open boiler room door
299.	TX	Dallas	SBS	1986	(Moore v. Polish Power, Inc.) 720 S.W.2d Tx App.	VOCs -- formaldehyde; trial court directed verdict for defendant; case remanded on appeal
300.	TX	Dallas	SBS, Legion	1991	J.L. Long Middle School	48 out of 70 teachers reported respiratory problems; one tested positive for Legionnaire's disease anti-bodies; 1934 heating and air conditioning systems are suspect; water fountains or air ventilation may be source
301.	TX	Houston	Legion	1982	Johnson Space Center - NASA	Possible outbreak; autopsy results pending
302.	TX	Houston	BRI	1991	Judwin Properties Apartment Complexes (Flores v. Winograd)	Exposure to misapplied chlordane; $10.5 million jury award
303.	TX	Houston	Legion	1992	Fire Station No. 1	Leaky roof, suspected dirty ventilation system tests pending
304.	TX	Wimberly	SBS	1988	Amelia Scudder Elementary School (Rogers v. Benjamin Moore & Co.)	Hydrocarbons, carbon dioxide and formaldehyde exposure from building materials; 40 plaintiffs, 25 defendants; trial set for spring 1993

305.	UT		Legion	1990	VA Hospital	
306.	UT	Moab	SBS	1987	State Social Services Office Building	Heating and cooling system, mold in carpet; NIOSH investigated and found no problems
307.	UT	Ogden	Legion	1991	IRS Service Center	Tests negative, water system purged
308.	VT	Burlington	Legion	1980	University of Vermont College of Medicine	Campus cooling tower; 17 dead, 62 ill
309.	VT	Burlington	Legion	1994	Brantwood Life Care Center	4 residents died; 10 patients are showing symptoms; source of bacteria in the nursing home has not been found
310.	VT	Montpelier	SBS	1992	Medical office	New carpet emissions; employees suffer from MCS; carpet was removed
311.	VA	Arlington	SBS	1988	USA Today Headquarters	Recent office renovation; NIOSH investigated tests inconclusive; 14 miscarriages
312.	VA	Arlington	SBS	1991	Kenmore Middle School	Illness due to poor ventilation; exposure to dust, chemicals, asbestos; 7 school teachers suffered miscarriages; NAS concluded: "Far too little is known about how such chemicals affect the young, and there is 'potential for concern.'"
313.	VA	Arlington	SBS	1993	Wilson High School	Sickness from formaldehyde
314.	VA	Blacksburg	SBS	1988	Cowgill Hall-Virginia Tech	Indoor air pollution; building was thoroughly cleaned; HVAC was upgraded
315.	VA	Chesterfield County	SBS	1992	Crestwood Elementary School	Tests inconclusive; 22 students ill
316.	VA	Clinton	SBS	1993	Francis T. Evans Elementary School	Broken air-flow valves; ventilation problem; students and teachers sick
317.	VA	Clinton	SBS	1993	Prince George's School	Headaches, fatigue, coughing and stuffy nose; ventilation problem
318.	VA	Gaithersburg	SBS	1991	Diamond Elementary School	High absenteeism from respiratory illness; malfunctioning ventilation system
319.	VA	McLean	SBS	1986	Franklin Sherman Elementary School	Poor ventilation; 2 rooms were sealed off
320.	VA	Richmond	SBS	1989	Montrose Elementary School	Mold-infested ventilation system; school newly renovated; school was closed following complaints; school board readying lawsuit
321.	VA	Richmond	BRI	1990	Office	Loose fiberglass in ventilation system
322.	VA	Springfield	SBS	1990	West Springfield High School	Tests inconclusive, renovations recently completed; cluster fainting; no cause ever

						found; in 1994 eight environmental studies conducted; no significant findings; parents and students remain angry
323.	VA	Suffolk	SBS, Legion	1991	Human Resources Building	Inadequate ventilation and indoor contaminants
324.	VA	Virginia Beach	SBS	1989	Salem High School	Inadequate HVAC system; high carbon dioxide levels; mold/mildew
325.	VA	Virginia Beach	SBS	1990	Birdneck Elementary School	Inadequate HVAC system
326.	VA	Virginia Beach	SBS	1990	Brandon Junior High School	Tests inconclusive; problem appeared after walls were painted
327.	WA	Asotin	SBS	1992	Asotin Elementary School	Students and teachers suffered headaches and nausea; glue may be source; were relocated until problem resolved; ventilation system needed adjustment
328.	WA	Coeur d'Alene	SBS	1988	Fire Station No. 2	Station located next to a landfill; fumes entering ventilation system; employees were evacuated to another work site
329.	WA	Everett	SBS	1981	North Middle School	Problems since 1981; school closed in 1994 because students vomited, suffered headaches and nausea; changed ventilation systems and filtering systems; committee was formed to determine baseline for IAQ and respond to future incidents; school will replace carpet with tile, and reassemble all parts of school's ventilation system; officials plan to evaluate fresh air entry into school
330.	WA	Kettle Falls	SBS	1990	Kettle Falls High School	Black mold; 25% absenteeism
331.	WA	Monroe	Legion	1987	Monroe Reformatory, Washington Dept. of Corrections	Bacteria in water supply
332.	WA	Pomeroy	SBS	1989	Pomeroy High School	4 staff suffering symptoms; ventilation, heating and cooling systems adjusted
333.	WA	Seattle	SBS	1980	State Department of General Administration	Poor air circulation; build-up of toxic fumes from building materials
334.	WA	Seattle	SBS	1991	Harbourview Medical Center	Low ceilings and inadequate ventilation
335.	WA	Seattle	SBS	1991	John Hay Elementary School	Inadequate ventilation; 24 ill
336.	WA	Seattle	SBS	1991	Snohomish County Courthouse	Poor ventilation, copy machines, damp carpeting

337.	WA	Seattle	SBS	1993	Port of Seattle Building	Employees complain of watery eyes, headaches, nausea and vomiting; improve ventilation; hired an environmental and engineering consultant
338.	WA	Spokane	Legion	1985	City Public Safety Building	Bacteria in air conditioning unit
339.	WA	Spokane	SBS	1992	Garry Middle School	Students experienced respiratory problems; source was never identified
340.	WA	Tacoma	SBS	1990	Highrise office building	
341.	WA	Tacoma	SBS	1992	King County Courthouse	IAQ tests pending
342.	WA		SBS		(Tarrant v. Zittings Reality) Wa. App. Case No. 10059-6-111	VOC - formaldehyde; ruled realtor has no duty to disclose
343.	WV	Welch	SBS	1989	Mount View High School	Tests revealed "tight building syndrome present"
344.	WI	Eau Claire	Legion	1979	Holiday Inn	Hotel found guilty of negligence in deaths of 3 guests; bacteria entered air conditioning system through open fireplace damper
345.	WI	Madison	SBS	1992	State Public Defender's Office Schmitt v. Bartow Associates	Worker's compensation award of $2,300; inadequate ventilation
346.	WI	Green Bay	Legion	1991	Brown County Mental Health Center	
347.	WI	Milwaukee	BRI	1990	West Milwaukee School District (Byrne v. West Allis/W. Milwaukee School District)	Airborne fungi; defense verdict
348.	WI	Milwaukee	SBS	1991	809 Building	HVAC system
349.	WI	Monona	SBS	1993	Nichols Elementary School	Student ill allegedly from carpet emissions; notice of claim filed with state attorney general seeking $1.2 million in damages
350.	WI	Port Washington	SBS	1989	Ozaukee County Courthouse	Ventilation systems, bacteria in air conditioning system, VOCs in carpets, exhaust fumes, ozone
351.	WI	Sheboygan	Legion	1986	Plastics plant	Cooling towers
352.	WI	Traux	SBS	1991	Madison Area Technical College	"Litany" of IAQ problems; innovative rooftop chute system installed

OSHA SEPARATES ETS FROM ITS PROPOSED RULE ON IAQ BY CLAIMING THAT VENTILATION PROCEDURES CANNOT ADDRESS ETS. OSHA'S POSITION IS GROUNDLESS, CONTRARY TO BOTH THEORY AND PRACTICE, AND DICTATED BY A POLICY DEDICATED TO THE ELIMINATION OF SMOKING

OSHA's Proposed Rule on IAQ addresses common indoor air contaminants such as microbiologicals, dusts, chemicals and by-products of human metabolism through provisions designed to ensure the proper operation and maintenance of ventilation systems. Increased ventilation, proper air distribution, and maintenance of a building's heating, ventilation and air-conditioning (HVAC) system effectively serve to dilute and remove such common substances from the indoor air. (59 FR 16003)

With remarkable disregard for submissions to the OSHA RFI docket, the published scientific literature and accepted theoretical and practical knowledge about HVAC design and operation, OSHA states that the ventilation approach in its Proposed Rule on IAQ cannot be applied to complaints about, or exposures to, ETS in the workplace. (59 FR 15970, 16003)

OSHA states that "the primary objective of the tobacco smoke provision is to eliminate the nonsmoker's exposure to ETS" [emphasis added]. (59 FR 16016) OSHA's Proposed Rule on indoor air quality in work environments, while presenting the outward appearance of a single, comprehensive approach to indoor air

quality problems through ventilation, actually constitutes two separate rulemakings -- one for environmental tobacco smoke, and one for every other potential constituent in indoor air. OSHA proposes two separate standards, one designed to reduce exposures to (and potential risks from) every potential substance occurring in indoor air (except ETS), and another designed to eliminate all exposure to ETS in the workplace. According to the Proposed Rule on ETS, the elimination of smoking from the workplace means that it is to be either prohibited entirely or restricted to a separate room that is negatively pressurized and exhausted immediately to the outdoors. (59 FR 16023)

OSHA's position on ETS is neither technically nor scientifically supported or supportable. OSHA states, but does not demonstrate, that ventilation parameters fail to effectively reduce and minimize exposures to ETS. (59 FR 15970, 16003) OSHA attempts to justify its position on the nonapplicability of ventilation to ETS with four unsubstantiated remarks in the Proposed Rule, namely:

(1) "Ventilation systems are designed only to remove occupant-generated contaminants, such as carbon dioxide and odors. These types of systems were not designed to dilute multiple point sources of contaminants that are typically found in non-industrial workplaces." (59 FR 15973) The statement is not referenced and is in direct contradiction with OSHA's own Proposed Rule for a

ventilation-based approach to IAQ problems (29 CFR § 1910.1033(d)(1));

(2) The Proposed Rule also states: "Natural and mechanical ventilation systems are designed primarily to limit the accumulation of products of human respiratory metabolism, and secondarily to limit odor; not to control the byproducts of biomass combustion. Thus, smoking indoors creates air pollution which is not adequately abated by customary ventilation systems." (59 FR 15986) This statement also is not referenced and is not supported by the scientific literature. (See analysis below);

(3) The Proposed Rule further states: "Dilution ventilation offers no protection in those cases where, due to the close proximity to a smoker (e.g., contaminant point source), the nonsmoking employee may be exposed to large amounts of sidestream and exhaled mainstream smoke (ETS). Due to the limitations of general ventilation, the smoke cannot be removed from the air before reaching the breathing zone of nearby employees." (59 FR 15991) The passage describes a situation in which a given ventilation system may "short-circuit", i.e., a case in which supply air is removed from the space before it can effectively dilute indoor air constituents. OSHA does not explain why such an occurrence would apply only to ETS and not to other constituents found in the indoor air. Moreover, OSHA does not represent in any

way that such situations are common, nor does it provide quantitation of the extent to which individuals are so exposed; and

(4) The Proposed Rule contends: "The carcinogenicity of ETS discounts the use of general ventilation as an engineering control for this contaminant." (59 FR 15991-2) The claim that ETS is a carcinogen and therefore cannot be addressed by general ventilation contradicts OSHA's own position on permissible exposure limits (PELs) for numerous so-called "carcinogens" in the workplace.

OSHA's Proposed Rule rejects application of the ventilation rates of an authoritative, national consensus standard on ventilation that was designed to address ETS and other IAQ constituents; the Standard, ASHRAE 62-1989, has been incorporated into the nation's major building codes (and is therefore incorporated by reference into OSHA's own Proposed Rule on IAQ); the Standard's effectiveness is supported in the published scientific literature and by IAQ field applications

The Proposed Rule recognizes ASHRAE Standard 62-1989 ("Ventilation for Acceptable Indoor Air Quality") as a "major ventilation guidance document available to HVAC practitioners," but rejects explicit application of the Standard's ventilation rates to ETS in workplace venues. The Proposed Rule states: "[I]t can only be inferred that the standard [ASHRAE 62-1989] was mostly based on satisfaction of sensory comfort rather than based on the control of

contaminants like ETS which may contribute to adverse health effects like lung cancer and heart disease." (59 FR 15992) However, the express purpose of ASHRAE Standard 62-1989 is to establish ventilation rates and procedures for various indoor settings in order to "control carbon dioxide and other contaminants with an adequate margin of safety and to account for variations among people, varied activity levels, and a moderate amount of smoking."[1] (Ex. 3-1074) The ASHRAE consensus Standard has been adopted by numerous states and by the major building code organizations in the United States. (Ex. 3-1074) It is the current design standard for ventilation systems in new, remodeled and renovated buildings in the U.S.

The precursor ventilation standard to ASHRAE Standard 62-1989, ASHRAE 62-1981, recommended two levels of ventilation, one for areas in which smoking was permitted, and another substantially lower rate for areas where smoking was prohibited. The prescribed ventilation rate in ASHRAE 62-1981 for offices in which smoking was permitted (20 cubic feet outside air per minute per person (cfm/person)) was 4 times greater than the rate recommended for nonsmoking areas (5 cfm/person).

IAQ problems were reported by those who followed the minimum (nonsmoking) ventilation rates specified in ASHRAE 62-1981. Research indicates that the ventilation rate for nonsmoking areas

(5 cfm/person) is insufficient to efficiently dilute carbon dioxide and body odor, and that occupants under such conditions may complain of "stuffy" air. Other research indicates that a ventilation rate of at least 15 cfm/person, the minimum rate recommended by ASHRAE Standard 62-1989 and three times the rate recommended for nonsmoking areas by ASHRAE 62-1981, would be necessary and sufficient to disperse normally occurring ambient substances (e.g. CO_2, body odors, etc.), as well as tobacco smoke.[2-4] (Ex. 3-440)

In recent testimony before a Congressional Subcommittee, Mr. John Janssen, the Chairman of the ASHRAE Project Committee for Ventilation Standard 62-1989, stated:

> Prior to the oil embargo of 1973, buildings tended to be over-ventilated and indoor air quality problems were not widely recognized. Ventilation recommendations for office spaces, for example, ranged all the way from 5 to 25 cubic feet per minute per occupant. In 1973, ASHRAE published the first edition of Standard 62, which allowed a minimum of 5 cfm of outdoor air per person for some applications. Most state and city building codes still reference ASHRAE Standard 62-1973, (Standard for Natural and Mechanical Ventilation.)
>
> In 1981, the ASHRAE Ventilation Standard was revised to incorporate new technology and new awareness of such issues as tobacco smoke, which was not mentioned in the 1973 version. Research results (some ASHRAE sponsored) showed that the minimum ventilating rate of 5 cfm per occupant permitted under 62-73 would not adequately control occupant-emitted odors. At least 15 cfm per occupant was needed to

> reduce the odor level to a point acceptable to 80 percent of the people entering an occupied space. This amount of ventilation was found sufficient to control tobacco-smoke odor when the smoking rate is about today's average.[5] (Ex. 3-1074)

In ASHRAE's submission to the RFI docket, Janssen writes:

> ANSI/ASHRAE Standard 62-1989 is an authoritative guide for achieving acceptable indoor air quality. The Standard defines ventilation rates needed to achieve freedom from odor, irritation and create a comfortable indoor environment. As the only nationwide consensus-based technical standard on ventilation and acceptable indoor air quality, ASHRAE recommends Standard 62-1989 as the standard of choice for adoption by reference for state and local building codes and regulations. (Ex. 3-440)

A number of respondents to the RFI endorsed the ventilation procedure specified in ASHRAE Standard 62-1989 and recommended its adoption as the basis to OSHA's Proposed Rule on IAQ. (Law Associates, Ex. 3-1200; ENV Services, Inc., Ex. 3-1089; Organization Resources Counselors, Inc., Ex. 3-1084; the National Environmental Development Association's Total Indoor Environmental Quality Coalition (NEDA/TIEQ), Ex. 3-1054; Healthy Buildings International (HBI), Ex. 3-1053; Systems Applications International (SAI), Ex. 3-1052; U.S. Navy, Ex. 3-982; Stellmack Air Conditioning and Refrigeration, Ex. 3-978; Oklahoma Dept. of Labor, Ex. 3-945; Pennsylvania AFL-CIO, Ex. 3-908B; Duke Power Company, Ex. 3-860; American Association of Occupational Health Nurses (AAOHN), Ex. 3-

803; American Federation of Government Employees, Ex. 3-529; ASHRAE, Ex. 3-440)

Other respondents, including governmental and private IAQ investigators and mitigation experts, industrial hygienists and engineers, recommended use of the Standard for achieving acceptable indoor air quality. (Meckler Engineers Group, Ex. 3-1081; Theodor D. Sterling & Associates (TDSA), Ex. 3-1073; Occupational Illness Support Group Local 12, Ex. 3-1017; R.J. Reynolds Tobacco Company (RJR), Ex. 3-1087; U.S. Navy, Ex. 3-982; Business Council on Indoor Air (BCIA), Ex. 3-933; Gershon Meckler Associates, Ex. 3-879; the Center for Environmental Assessment, Inc., Ex. 3-687; Sheet Metal and Air Conditioning Contractor's National Association, Inc. (SMACNA), Ex. 3-856; International Brotherhood of Teamsters, Ex. 3-858; Consolidated Edison Company of New York, Ex. 3-828; Caterpillar, Inc., Ex. 3-805; Philip Morris Companies, Ex. 3-1074; United Technologies, Ex. 3-651; Dow Chemical Company, Ex. 3-502; Thomas E. Glavinich, D.E., P.E., Ex. 3-498; Ford Motor Company, Ex.3-447; Systems Applications International (SAI), Ex. 3-1052)

One of the largest private IAQ diagnostic and mitigation firms in the U.S., Healthy Buildings International, Inc., writes that "ASHRAE Standard 62-1989, 'Ventilation for Acceptable Air Quality,' is perhaps the single most useful document we have in our efforts to communicate the proper practices for ensuring good

indoor air quality in commercial buildings. We support its use, continued development and incorporation into future building codes, standards and IAQ legislation." (Ex. 3-1053)

The submission by Meckler Engineers Group states:

> ASHRAE Standard 62-1989 is the only recognized authority that specifies the desired performance of building ventilation systems. . . . If OSHA decides that it is appropriate to regulate workplace IAQ at the national level, adoption of ASHRAE Standard 62-1989 would be the best strategy. (Ex. 3-1081)

The EPA's submission to OSHA includes its handbook for remediation of indoor air quality problems. EPA's recommendations include: "Compare design air quantities to building codes for the current occupancy or ventilation guidelines (e.g., ASHRAE 62-1989), and compare ventilation rates to ASHRAE 62-1989." The handbook also recommends that it would be "informative to see how your ventilation rate compares to ASHRAE 62-1989, because that guideline was developed with the goal of preventing IAQ problems." (Ex. 3-1075 D)

The ventilation rates and design specifications of ASHRAE 62-1989 are effective in the mitigation of indoor air quality problems

In a 1991 publication, Thompson et al., described various retrofit projects for HVAC systems in 26 schools across the U.S.

The retrofits followed the design specifications set forth in ASHRAE Standard 62-1989 for ventilation.[6] The retrofits were, according to the authors, "very effective" at reducing radon and CO_2 levels in the schools. The authors also reported that "many of the occupants" believed that IAQ had improved. (Ex. 3-1074)

The State of Wisconsin Safety and Building Division reports that, in their experience:

> Almost without fail, . . . complaints are resolved by ventilating the offices or classrooms per state code. This is a provision of outside air (up to 20 cubic feet per minute) per person. . . . These ventilation requirements are supported by the American Society of Heating, Air-Conditioning, and Refrigerating Engineers (ASHRAE). (Ex. 3-10)

Downing and Bayer recently reported results from more than 35 building IAQ investigations.[7] They reported that the most "common source of IAQ problems has been the lack of proper operation and maintenance (O&M) of buildings." They further observed that "in more than 80 percent of the investigations to date, changes in building O&M significantly improve the perception of IAQ by the occupants." The researchers recommend, as part of their O&M procedure check list for indoor air quality, "to raise outdoor air ventilation to ASHRAE recommended minimums." (Ex. 3-1074)

In 1991, investigators of the U.S. EPA's headquarters in Washington, D.C., reported that they were "unable to establish consistent relationships between major environmental parameters and self-reported health symptoms among the sampled employees."[8] However, based on the number and frequency of complaints among workers in those buildings, the research group recommended that "an attempt to maintain indoor environment in accordance with the ASHRAE guidelines should be made." (Ex. 3-1074)

The ventilation rates in ASHRAE Standard 62-1989 specifically address ETS and have been proven effective in the dilution and removal of ETS constituents and the minimization of exposure to nonsmokers

The effectiveness of ASHRAE Standard 62-1989 for the dilution of ETS constituents has been evaluated in a number of scientific studies that were submitted to the OSHA RFI docket.

In 1990, researchers presented results of their work comparing the effects of increased ventilation recommended by ASHRAE 62-1989 in areas where smoking is permitted and in areas where it is prohibited. Through the aid of computer models, the researchers demonstrated that air quality in the areas where smoking is permitted does not differ significantly from air quality in nonsmoking areas, where both areas are supplied with outdoor air at levels recommended by ASHRAE 62-1989.[9] (Ex. 3-1065) For

comparison, OSHA's PEL for nicotine is 100 times greater (0.5 mg/m^3). (29 CFR 1910.1000, Table Z-1).

In their review of ETS-related air quality monitoring in different workplaces under various smoking conditions, researchers from TDSA Ltd. conclude "in office areas in which (a) smoking is allowed, and (b) outside air ventilation rates meet or exceed the ASHRAE ventilation standard, nicotine concentrations have typically been less than 5 ug/m^3 and respirable suspended particle levels have ranged between 20 ug/m^3 and 60 ug/m^3." (Ex. 3-1073)

In their submission to the OSHA docket, scientists from HBI, Inc. summarized the results of a paper entitled "The Measurement of Environmental Tobacco Smoke in 585 Office Environments." (Ex. 3-1053) The authors write:

> Computer analysis shows that when 'blindfolded' for presence or absence of smokers, in most cases realistic smoking levels do not significantly influence the aspects of air quality that were measured, and spill over from smoking areas into nonsmoking areas appears to minimal. This work further reinforces the position the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) has taken on ETS in office buildings in ASHRAE Standard 62-1989 (1989), that acceptable air quality can be maintained in properly ventilated offices with a moderate amount of smoking, even without smoker segregation.

Professor Alan Hedge offers the following observation on the basis of his extensive experience in monitoring ETS constituents during investigations of sick-building syndrome: "Our data show that modern ventilation systems are capable of diluting the small pollutant loads from smoking at the levels which we observe, without necessarily exposing nonsmokers to significant elevated levels of indoor air pollutants." (Ex. 3-955)

Company scientists from R.J. Reynolds reported on a recently completed study of four office buildings. (Ex. 3-1087) Two of the buildings investigated had a policy of unrestricted smoking; in the other two buildings, smoking was restricted to separately exhausted lounges. Regardless of smoking policy, RJR reports all ventilation and indoor air quality indicators were "well within applicable standards." The authors write:

> In summary this study demonstrates conclusively (a) that with an HVAC system that is adequately designed, operated in accordance with current ASHRAE standards and properly maintained, all indicators for ETS are at extremely low, de minimis levels, even in the presence of substantial smoking activity, and (b) that such smoking activity has a negligible effect on contaminant levels in buildings where smoking is unrestricted. (Ex. 3-1087)

RJR concludes:

> RJR believes, based on its own detailed research and the consistent results of other

> workplace assessments, that a properly designed and maintained HVAC system that is operated in accordance with the ventilation rate procedures of the ASHRAE Standard 62-1989, will be effective in assuring that exposures to ETS will be de minimis.

Based on their own case studies, the National Energy Management Institute (NEMI) acknowledges that exposure "to the odor caused by excessively high concentrations of ETS can be annoying to nonsmokers." NEMI suggests that "several avenues exist to address this problem. First and foremost is to apply the ASHRAE 62-1989 ventilation standard . . . [W]orkplaces operating in accordance with ASHRAE 62-1989 will not have ETS annoyance problems because the ventilation system will effectively remove all smoke." (Ex. 3-1183)

In their comprehensive review of indoor air quality in non-industrial occupational environments, Morey and Singh write that "ASHRAE Standard 62-1989 is probably the most important document in the IAQ literature."[10] (Ex. 3-505) They note:

> It reflects a consensus reached since 1983 by knowledgeable individuals from engineering, industrial and academic groups. Janssen points out that the ventilation rates recommended in Standard 62-1989 for the most part are similar to 'recommended' rates in Standard 62-1973 and to the rates recommended for smoking environments in Standard 62-1981.
>
> A key feature of Standard 62-1989 and its ventilation rate procedure is the increase in the minimum outdoor ventilation rate from 5 to

15 cfm per person. Outdoor air requirements recommended by the ventilation rate procedure make no distinction between 'smoking-allowed' and 'smoking-prohibited' areas. A minimum of 15 cfm of outdoor air per person as specified in the ventilation rate procedure is recommended because new research indicated that this is the minimum amount of outdoor air needed to dilute body and tobacco smoke odors to acceptable levels. The outdoor air requirements specified in the ventilation rate procedure must be delivered to the occupied zone. Design assumptions with regard to ventilation rates and air distribution to the occupied zone are required by Standard 62-1989.

Standard 62-1989 also requires that the design documentation for a HVAC system state clearly which assumptions are used in design. This allows others to estimate the limits of the HVAC system in removing air contaminants prior to commissioning and prior to the introduction of new contaminant sources into the occupied space.

A key provision in Standard 62-1989 now requires that when the supply of air to the occupied zone is reduced (for example, in VAV systems), provision be made to maintain minimum flow rates of outdoor air throughout the occupied zone.

Summary

OSHA's contention that ETS cannot be addressed through ventilation parameters (or indeed through its own Proposed Rule for IAQ and the proper operation and maintenance of ventilation systems) is not supported in the scientific and technical literature. OSHA's position contradicts well-accepted practice that demonstrates the effectiveness of ventilation parameters in

addressing complaints about, and exposures to, ETS. A workplace that is ventilated according to OSHA's Proposed Rule on IAQ, i.e., for the reduction of levels of indoor substances associated with sick-building syndrome complaints, will also significantly dilute constituents of, and reduce and minimize exposures to, ETS. Case reports and published scientific studies support this position.

OSHA's refusal to endorse ASHRAE Standard 62-1989's ventilation rate procedures contradicts the specifications in its own Proposed Rule on IAQ, i.e., "that employers maintain and operate the HVAC system to provide at least the minimum outdoor air ventilation rate . . . required by the applicable building code . . . in effect at the time the facility was constructed, renovated and remodeled" [emphasis added]. (59 FR 16026) ASHRAE Standard 62-1989 has served as the ventilation criteria document for new and renovated construction in the U.S. since 1990. (It is therefore part of OSHA's own Proposed Rule on IAQ).

OSHA's denial that a general, ventilation-based standard for IAQ is also applicable to ETS forces OSHA to an unsupported, untenable, contradictory and potentially unethical position, in that it would allow smokers to be exposed to their own ETS in the workplace. Virtually every substance in the indoor air imputed to ETS by OSHA is also generated by other sources. In effect, OSHA argues that substance "X" can be addressed effectively through

ventilation, unless precisely the same substance at precisely the same exposure level originates from ETS. (59 FR 15979-80, 15984, 15985, 15987-88) OSHA obviously has no basis for this position.

The separate treatment of IAQ and ETS permits OSHA to give the appearance of satisfying the technological/economic feasibility argument for its ETS standard. A ban on smoking in the workplace, OSHA argues, will cost nothing, and the provision of a separately ventilated smoking area is "an option, not a requirement, under the proposed regulation." (59 FR 16013) On the other hand, if ETS were included among the ventilation-based provisions of OSHA's IAQ standard, the technological/economic feasibility of addressing ETS would become part of the overall IAQ standard, now estimated by OSHA to cost approximately $8 billion. The technical and economic feasibility of dealing with ETS would then stand or fall with the entire IAQ standard, something OSHA appears unwilling to risk in its quest to completely "eliminate the nonsmoker's exposure to ETS." (59 FR 16016)

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SECTION IV

ETS IN THE WORKPLACE:

CHARACTERIZATION AND EXPOSURE

ETS IN THE WORKPLACE:
CHARACTERIZATION AND EXPOSURE

1. Introduction and Overview

OSHA FAILS TO DEMONSTRATE A SIGNIFICANT RISK OF MATERIAL IMPAIRMENT FOR ANY DISEASE ENDPOINT AMONG NONSMOKERS REPORTEDLY EXPOSED TO ETS IN THE WORKPLACE. PARTICULARLY, OSHA FAILS TO ESTABLISH THAT EXISTING EXPOSURES TO ETS IN THE WORKPLACE POSE A SIGNIFICANT RISK OF MATERIAL IMPAIRMENT TO THE HEALTH OF NONSMOKERS

OSHA's Proposed Rule states that "before the Secretary can promulgate any permanent health or safety standard, he must find that a significant risk of harm is present in the workplace and that the new standard is reasonably necessary to reduce or eliminate that risk. Industrial Union Department, AFL-CIO v. American Petroleum Institute, 444 U.S. 607, 639-642 (1980) (Benzene)." (59 FR 16000) In an attempt to establish significant risk for ETS, the Proposed Rule reviews various disease endpoints reportedly associated with ETS, including (i) irritation, (ii) pulmonary effects (other than cancer), (iii) cardiovascular disease, (iv) reproductive effects and (v) lung cancer. In addition, characteristics of mainstream smoke (the smoke to which the smoker is exposed) and sidestream smoke (the smoke from the burning tip of the cigarette) are enumerated, and a select number of studies on ambient exposures to ETS constituents in the workplace are examined. Risk analyses for lung cancer and cardiovascular disease among nonsmokers in the workplace are

presented. The risk analyses for lung cancer and heart disease are each based upon single epidemiologic studies that are then applied to estimates of mortality for the total nonsmoking workforce in the U.S.

OSHA fails to demonstrate a significant risk of material impairment for any disease endpoint among nonsmokers reportedly exposed to ETS in the workplace. Particularly, OSHA fails to establish that existing exposures to ETS in the workplace pose a significant risk of material impairment to the health of nonsmokers. Failure to establish significant risk at current exposure levels is precisely the reason for which the U.S. Court of Appeals in 1992 vacated OSHA's Air Contaminants Standard. (American Federation of Labor and Congress of Industrial Organizations v. Occupational Safety and Health Administration, 965 F.2d 962 at 20) Moreover, in 1991 OSHA acknowledged that it "does not have adequate information on the current level of exposure to ambient tobacco smoke in workplaces to accurately assess the existing level of occupational risk" [emphasis added]. (Secretary's Response at 6, ASH v. OSHA) The data cited in OSHA's Proposed Rule do not remedy that deficiency.

No measured ambient exposure data for ETS are included in OSHA's estimate of risk due to ETS in the workplace

OSHA's analysis of "significant risk" for ETS is based upon two epidemiologic studies in which ETS exposures are not measured directly. ETS exposures were estimated in the two studies by individual recall of exposure. Accurate quantitative measures of exposure cannot be ascertained through those kinds of studies. **No measured ambient exposure data for ETS are included in OSHA's estimate of risk due to ETS in the workplace.** Thus, OSHA offers an analysis of risk and a proposal for the complete elimination of ETS in the workplace **without reference to any measured exposure data.**

However, studies that report measured data for constituents of ETS in the air of indoor work environments are currently available. OSHA's discussion of such studies is limited, selective and unrepresentative of current ETS exposure data. Indeed, OSHA's Proposed Rule does not reference a single ambient air monitoring study for the workplace published after 1991, the same year in which OSHA admitted that it had no adequate data on "current levels of exposure" for ETS. (59 FR 15990-1)

The Proposed Rule states that "estimating the risk from exposure to ETS requires the use of some measure of the extent of exposure." (59 FR 15997) OSHA acknowledges failure to integrate

ambient ETS exposure data into its analysis of significant risk and states that "since there is no definition of, nor an established method for quantifying, exposure, it is not possible to determine a 'dose limit' that would eliminate significant risks." (59 FR 16001) As will be demonstrated in the analysis that follows, current exposure data on ETS levels in the workplace do not support the conclusion that ETS poses a "significant risk" to nonsmoking workers.

The Occupational Safety and Health Act of 1970 requires the Secretary of Labor to develop a standard "that most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity" [emphasis added]. (59 FR 16007-8) While the interpretation of what constitutes "best available evidence" may be an open question, it is incumbent upon OSHA to at least consider available data on workplace exposures to ETS, as well as all available data on health effects that are associated specifically with workplace exposures to ETS. OSHA's Proposed Rule does neither. It uncritically accepts, without discussion of quality or obvious weaknesses, one epidemiologic study on ETS to determine "significant risk" of lung cancer in the workplace. (59 FR 15995) The data from 13 other available studies on nonsmoker lung cancer in the workplace, eight of which are from the U.S, are ignored. The Proposed Rule also utilizes a single epidemiologic

study on cardiovascular disease (CVD) that deals with spousal smoking in the home to generate "significant risk," while ignoring available studies on reported ETS exposures and CVD in the workplace. (59 FR 15995) The Proposed Rule bases its "exposure assessment" for ETS upon a select, outdated and unrepresentative group of ambient air monitoring studies, some of which do not deal with workplace exposures to ETS at all. (59 FR 15990-2) OSHA's analyses will be discussed in the following sections.

OSHA's discussion of tobacco smoke constituents does not establish that ETS is carcinogenic

OSHA's Proposed Rule on general IAQ does not include ETS because, according to OSHA, "the carcinogenicity of ETS discounts the use of general ventilation as an engineering control for this contaminant." (59 FR 15992) Nothing in the OSH Act provides any support for such a policy. It is essentially the same as the "lowest feasible level" policy the Supreme Court struck down in Benzene. In addition, OSHA's attempt to demonstrate the "carcinogenicity" of ETS takes the form of an argument from analogy from mainstream smoke (the smoke to which the active smoker is exposed) to ETS. OSHA claims that ETS is chemically similar to mainstream smoke, and that "carcinogens" reportedly identified in mainstream smoke are also present in ETS. (59 FR 15979)

The Proposed Rule presents a table of substances labeled "Table II-2: 43 Chemical Compounds Identified in Tobacco Smoke for Which There Is 'Sufficient Evidence' of Carcinogenicity in Humans or Animals." (59 FR 15979-80) The 43 compounds listed therein are neither discussed nor reviewed in the Proposed Rule. Moreover, the Proposed Rule concedes that:

(1) Few of the substances identified as constituents of tobacco smoke (in the "list of 43") have been identified in ETS, i.e., "few of these individual [inhalational] constituents [imputed to ETS] have been identified and characterized." (59 FR 15987);

(2) The substances listed are also derived from other sources, i.e., "most of the identified carcinogenic components [in the list of 43] are not unique to ETS." (59 FR 15998); and

(3) Individual substances responsible for the alleged "carcinogenicity" of tobacco smoke have never been identified, i.e., "the mechanism of carcinogenicity from exposure to ETS is not known." (59 FR 15998)

The so-called list of "43 carcinogens in tobacco smoke" has been challenged in the scientific literature.[1-4] One commentator notes:

> Many of these 43 MS [mainstream smoke] and/or tobacco components should be excluded from the list on the basis of published data on their tumorigenicity (or lack of it) in laboratory animals at levels determined in MS, their lack of tumorigenicity in most instances on inhalation, and the equivocal evidence of their tumorigenicity in humans at levels in MS. . . .
>
> . . . Only five of the 43 components have produced respiratory tract tumors in laboratory animals exposed to the component via inhalation. Many have never been tested in an inhalation system. One component of great interest, 'benzo[a]pyrene,' has only produced lung carcinoma via inhalation in animals at an extraordinary massive dose, a finding classified as 'equivocal.' (RTECS, 1987).[1]

One of the few substances on the "list of 43" that has been tested via animal inhalation, nickel, has reportedly generated human-type lung cancers.[1] Nickel is derived from a number of sources. It was tested for carcinogenicity at exposure levels far in excess of those found in the workplace, the environment or in tobacco smoke. For example, it has been estimated that 40,000 cigarettes burning simultaneously in a one hundred cubic meter enclosure would be required in order to generate a level of exposure to nickel equivalent to the Permissible Exposure Limit (PEL) established by OSHA.[2-4]

The "list of 43" constituents was developed in the 1970s, and it has been suggested that "many of the listed components are not relevant to those that would be found if the analysis were

conducted on more recent cigarettes."[1] This is particularly true of the substances dibenz(a,h)acridine and dibenz(a,h)anthracene. N'-nitrosonornicotine appears to be the only constituent characteristic of tobacco smoke from the "list of 43"; most of the constituents on the list, although theoretically identifiable, have never been identified in ETS (e.g., arsenic, chromium, etc.).[3]

Most of the substances were tested for carcinogenicity in animals through means other than inhalation. Skin painting of several components reportedly resulted in skin tumors, and neoplasms were reported in animals through ingestion and injection of various constituents. Several of the substances, e.g., benzene, formaldehyde and nitrosodiethylamine, reportedly produced neoplasms in areas other than the lung.[3] (Epidemiologic studies of workers report increased risks of leukemia by benzene; liver cancer by vinyl chloride; and urinary bladder cancer by 4-aminobiphenyl and 2-naphthylamine.)

Neither nicotine nor cotinine, the two substances selected by OSHA as biologic markers for exposure to ETS, are indicators of exposure to any of the constituents on the "list of 43." (59 FR 15974, 15991, 15998) Neither substance is itself a "suspect carcinogen."[5] OSHA concedes that, since the constituents on the list of 43 are not unique to ETS, "direct measurement of the carcinogenic components or related biomarkers in biological fluids

would not provide a unique measure of exposure from ETS." (59 FR 15998) OSHA explicitly rejects one group of nine constituents on the "list of 43," the polycyclic aromatic hydrocarbons (PAHs), as candidate biomarkers precisely because they are found in low concentrations in tobacco smoke and because they are not unique to ETS. (59 FR 15998)

Mainstream and sidestream smoke comparisons are not relevant to ETS

The Proposed Rule presents two additional tables of constituents ascribed to tobacco smoke. (59 FR 15987-8) The tables provide a comparison of the amount of each constituent reported in mainstream smoke with the amount reported for the constituent in sidestream smoke (the smoke directly at the burning tip of a cigarette). OSHA's argument is that if constituent levels in sidestream and mainstream smoke are compared, quantities are greater in sidestream smoke. However, the relevance of these comparisons to ETS is questionable.

A number of scientists have reported that ETS is not the same as either mainstream or sidestream smoke.[1,6-9] ETS is an aged and diluted mixture of sidestream and exhaled mainstream smoke; ETS is a dynamic, ever-changing mixture that undergoes chemical transformations and physical changes as it ages and is diluted in the air.[1,6-7] As one researcher in tobacco smoke chemistry has

observed, "there are profound physical and quantitative chemical differences" among the three kinds of smoke (mainstream smoke, sidestream smoke and ETS).[1] There are differences in physical and chemical properties and in relative concentrations of constituents. Studies indicate that constituents in ETS are hundreds to thousands of times more dilute than either sidestream or mainstream smoke.[8-9] Concentrations of ETS constituents in real-life situations are often below the limits of detection and measurement for even the most sensitive air monitors. Often, the contributions of ETS constituents to the ambient air are indistinguishable from background levels of the same constituents generated by other sources.[8]

OSHA's strategy of comparing mainstream and sidestream smoke ignores the profound effect of dilution in the ambient air upon tobacco smoke constituents. As two tobacco smoke chemists report:

> The important question is not the ratio of sidestream/mainstream but rather what is the concentration of the constituent in the indoor environment and how does it compare to levels from sources other than ETS. Studies based solely on observations of fresh sidestream, are highly and unrealistically concentrated ETS should take into account the possible differences between these smokes and ETS found in real life situations.[6]

Similarly, the 1986 Report of the Surgeon General notes: "SS [sidestream smoke] characteristics, as measured in chambers, do not represent those of ETS, as inhaled by the nonsmoker under non-experimental conditions."[10]

OSHA's tables of tobacco smoke constituents do not present levels reported for ETS. The relevance of OSHA's mainstream/sidestream smoke comparisons to workplace exposure levels of ETS is therefore questionable.

Elimination of ETS exposures in the workplace will not eliminate nonsmoker exposure to carcinogens; carcinogens imputed to ETS by OSHA have permissible exposure limits, yet OSHA establishes a "no-exposure" standard for ETS; OSHA's contradictory and insupportable position on ETS does not comply with OSHA's regulatory framework and undermines its own regulatory position on permissible exposure limits

OSHA's Proposed Rule acknowledges that the ordinary air in the non-industrial workplace contains substances that have been identified as "carcinogens," whether or not smoking takes place. (59 FR 15983, 15998) The Proposed Rule presents a list of "typical pollutants" emitted from building materials, furnishings, appliances, office equipment and supplies. (59 FR 15984) Many of the same constituents contained in the Proposed Rule's list of "tobacco smoke constituents" are found in OSHA's tables for emissions from office furnishings and materials. (59 FR 15987-8)

(See Table I) Benzene, for example, is emitted by paints, wood preservatives, cleansers, disinfectants, fuels, solvents, etc. Formaldehyde off-gases from particle board, pressed wood, resins, paneling, carpeting, upholstery, disinfectants, etc. Toluene is found in paints and other solvents, aerosol sprays, cleaners and disinfectants, air fresheners, fuels, etc. Benzo(a)pyrene is emitted from gas ranges, dryers, water heaters, and other combustion sources. Acetaldehyde, a substance in tobacco smoke described by OSHA as an "irritant," is found in adhesives, cosmetics, office supplies, and emanates from other combustion sources. It also is an organic effluent that is emitted naturally by humans through metabolism.[11]

OSHA's Proposed Rule therefore seeks to <u>eliminate</u> exposure to substances if they are derived from ETS, while seeking only to <u>reduce</u> levels of precisely the same substances if they are derived from sources other than tobacco smoke. (59 FR 16016, 16023-16027) Complete elimination of ETS will not eliminate exposure to substances imputed to it. OSHA cannot argue that the contribution of ETS to substances in the air of workplaces is "different" from, or greater than, contributions from other sources because OSHA does not present data on either ETS constituent levels or levels of constituents from sources other than ETS. Indeed, the Proposed Rule requests data "on the levels of these contaminants in non-industrial workplaces." (59 FR 15985)

TABLE I

Tobacco Smoke Constituents Also Found in Emissions From Other Indoor Sources*

Emitted Pollutants	Indoor Sources
Acetaldehyde	Human Metabolism Preprinted Paper Forms
Acetic Acid	Preprinted Paper Forms
Acetone	Preprinted Paper Forms Typewriter Corrections Fluid
Acrolein	Preprinted Paper Forms
Ammonia	Electrophotographic Printers, Photocopiers & Related Supplies Microfiche Developers/ Blueprint Machines
Benzene	Adhesives Caulking Compounds Clipboard/Particle Board Floor and Wall Coverings Paint, Stains & Varnishes Electrophotographic Printers, Photocopiers & Related Supplies
Carbon Monoxide	Appliances Cooking Heating
Formaldehyde	Adhesives Caulking Compounds Carpeting Ceiling Tiles Clipboard/Particle Board Floor and Wall Coverings Paints, Stains & Varnishes Carbonless Copy Paper
Phenol	Computer/Video Display Terminals
Polyaromatic Hydrocarbons	Appliances

Styrene	Carpeting Electrophotographic Printers, Photocopiers & Related Supplies
Toluene	Adhesives Clipboard/Particle Board Paints, Stains & Varnishes Computer/Video Display Terminals Electrophotographic Printers, Photocopiers & Related Supplies
Zinc stearate combustion products	Electrophotographic Printers, Photocopiers & Related Supplies

* Source: Proposed Rules, Federal Register, Vol. 59, No. 65, April 5, 1994, p. 15984, Table III-1 and Table III-2.

The elimination of ETS in the workplace cannot be achieved under OSHA's own regulatory framework. OSHA argues that tobacco smoke contains carcinogens and that no exposure to it should be permitted.* The list of constituents imputed to ETS by OSHA (59 FR 15979-80) contains suspected carcinogens currently regulated by OSHA through permissible exposure limits (PELs) (e.g., arsenic, benzene, chromium, nickel, formaldehyde, benzo(a)pyrene, etc.). (See Tables II-IV) OSHA cannot use substances with permissible exposures to demonstrate the "carcinogenicity" of ETS, and, at the same time, argue for its elimination.**

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*. Yet OSHA sets a Permissible Exposure Limit for airborne asbestos fibers at 0.2 fibers per cubic centimeter of air. Asbestos exposures permitted by OSHA could conceivably attain levels equivalent to millions of fibers in the air of offices, public places or other workplace venues. Asbestos, of course, is a Group A carcinogen that is not derived from ETS. Other designated carcinogens, such as benzene, chloroform, radon, styrene, carbon tetrachloride, etc. are also found in non-industrial buildings in the complete absence of ETS.

**. The Proposed Rule states that permissible exposure limits cannot be applied to ETS because (1) non-industrial environments do not have "administrative and engineering controls" of the industrial workplace (59 FR 15973); (2) OSHA cannot quantify ETS exposure and therefore cannot set an exposure limit "that would eliminate significant risk" (59 FR 16001); and (3) procedures for the identification of carcinogens "may not allow for the level of public input and policy review that is appropriate for this rulemaking . . ." (59 FR 16001) What OSHA really means is that many of the constituents imputed to ETS already have existing permissible exposure limits, set at levels tens to thousands of times greater than any level contributed by ETS.[2-4, 8] Were OSHA to apply a PEL to ETS, its entire regulatory structure would collapse; a PEL for ETS would necessitate lowering PELs for individual constituents to conform with potential ETS exposure levels.

TABLE II

Existing OSHA Permissible Exposure Limits
For Chemical Compounds Also Found in Tobacco Smoke*

Substance	ppm	mg/m^3
Acetaldehyde	200	360
Acrylonitrile; see 1910.1045	2 10 (ceiling limit)	
Arsenic, inorganic compounds (as As); see 1910.1018		0.01
Arsenic, organic compounds (as As)		0.5
Benzene; see 1910.1028	1	
Benzene; see Table Z-2 for the limits applicable in the operations or sectors excluded in 1910.1028[d]	10 25 (ceiling limit)	
Benzo(a)pyrene; see Coal tar pitch volatiles		
Cadmium (as Cd); see 1910.1027		0.005
Coal tar pitch volatiles (benzene soluble fraction), anthracene, BaP, phenanthrene, acridine, chrysene, pyrene		0.2
1,1-Dimethylhydrazine	0.5	1
Formaldehyde; see 1910.1048	0.75	
Hydrazine	1	1.3
Lead, Inorganic (as Pb); see 1910.1025		0.05
Nickel, metal and Insoluble compounds (as NI)		1
Nickel, soluble compounds (as NI)		1
2-Nitropropane	25	90
Styrene; see Table Z-2	100 200 (ceiling limit)	
Vinyl chloride; see 1910.1017	1	

* Source: 29 CFR § 1910.1000 Table Z-1, unless otherwise noted.

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TABLE III

Existing OSHA Permissible Exposure Limits
For Particulate Phase Constituents of Tobacco Smoke*

Substance	ppm	mg/m^3
Aniline and homologs	5	19
Benzo(a)pyrene; see Coal tar pitch volatiles		
Cadmium (as Cd); see 1910.1027		0.005
Coal tar pitch volatiles (benzene soluble fraction), anthracene, BaP, phenanthrene, acridine, chrysene, pyrene		0.2
Hydroquinone		2
Nickel, metal and insoluble compounds (as NI)		1
Nickel, soluble compounds (as NI)		1
Nicotine**		0.5
Phenol	5	19
Zinc oxide Respirable fraction		5
Zinc stearate Respirable fraction		5

* Source: 29 CFR § 1910.1000 Table Z-1, unless otherwise noted.
** Nicotine is in the gas phase of ETS.

TABLE IV

Existing OSHA Permissible Exposure Limits
For Vapor Phase Constituents of Tobacco Smoke*

Substance	ppm	mg/m³
Acetic acid	10	25
Acetone	1000	2400
Acrolein	0.1	0.25
Ammonia	50	35
Benzene; see 1910.1028	1	
Benzene; see Table Z-2 for the limits applicable in the operations or sectors excluded in 1910.1028[d]	10 25 (ceiling limit)	
Butadiene (1,3-Butadiene)	1000	2200
Carbon dioxide	5000	9000
Carbon monoxide	50	55
Dimethylamine	10	18
Formaldehyde; see 1910.1048	0.75	
Formic acid	5	9
Hydrazine	1	1.3
Hydrogen cyanide	10	11
Methylamine	10	12
Methyl chloride; see Table Z-2	500 1000 (ceiling limit)	
Pyridine	5	15
Toluene	200 300 (ceiling limit)	

* Source: 29 CFR § 1910.1000 Table Z-1, unless otherwise noted.

For the same reasons, OSHA cannot restrict its IAQ proposal to only the non-industrial workplace, while applying its de facto smoking ban to all workplaces, including both industrial and non-industrial venues. For the industrial worker, OSHA's plan affords no protection from ordinary indoor air quality constituents found in the work environment. This means that the nonsmoking industrial worker, under OSHA's Proposed Rule, may be exposed to levels of chemicals 100 to 1,000 times greater than those found in either the non-industrial workplace or in ETS while, at the same time, the worker is not to be exposed to even a single wisp of tobacco smoke.*** This position is ludicrous and clearly displays OSHA's policy objective for the elimination of smoking at all costs. OSHA's own regulatory framework in fact prohibits the kind of policy represented in its Proposed Rule on ETS.

***. Thus, Ford Motor Company's submission to the OSHA RFI docket states: "For the most part, PTS/ETS and IAQ issues are not associated with significant air contaminant concentrations. In resolving this issue, government and management come head-on into a basic philosophical dilemma of should there be one set of air contaminant limits for the office and one for the shop floor. If one subscribes to the theory that PTS/ETS in the concentrations normally found in offices is carcinogenic, then one would also have to conclude that most workers in factories and garages are exposed to harmful levels of carcinogens. Even conservative epidemiological studies suggest that this type of epidemic does not exist in the American workplace." (Exs. 3-447, 3-433)

OSHA's examination of workplace exposure studies on ETS is selective; the studies are outdated and are not representative of current ETS exposures in the workplace; the data from the Proposed Rule's discussion of ETS workplace exposures are not used in the determination of "significant risk" from ETS

OSHA's analysis of the data "available for . . . assessing exposure to ETS in the workplace" purports to establish average indoor levels of ETS and the percentage of average daily ETS exposure for nonsmokers in the workplace. (59 FR 15987-91) The Proposed Rule discusses a select number of studies that measure nicotine and respirable suspended particles as markers for ETS in the air of workplaces. The studies are used to determine a range of average exposures to ETS. Four "human activity pattern studies" are used to establish OSHA's claim that the "workplace is a major location of ETS-exposure to nonsmokers." (59 FR 15990)

The studies used in the Proposed Rule's discussion of ETS exposures are neither representative of, nor generalizable to, current levels of nonsmoker exposure in the workplace.

Human activity pattern studies for assessing workplace exposures to ETS are misinterpreted in the Proposed Rule; such studies are inherently inaccurate and not representative of current workplace exposure patterns

Human activity pattern studies are based upon an individual's recall of ETS exposure over a given period of time. Estimates of exposure are acquired through questionnaire responses and diary entries of study participants. Both the accuracy and the quantifiability of such "data," when compared to actual measures of ETS constituents in the air, have been challenged.[12-16] (e.g., Ex. 8-66, Coultas, et al.) Two of the four human activity pattern studies cited in the Proposed Rule were conducted in the mid-1980s. Data for the third cited study were gathered in 1990 and a fourth study cited by the Proposed Rule is undated. (59 FR 15988-90) The generalizability of the sample populations in each of the respective studies to the entire U.S. workforce under current exposure conditions is not addressed in the Proposed Rule.

The first study cited in the Proposed Rule is the California Activity Pattern Survey (CAPS), conducted in 1987-88. (59 FR 15989) The Proposed Rule suggests that the California study indicates that "the most powerful predictor of potential exposure to ETS" is the workplace. OSHA claims that the study establishes that "77 percent of males and 85 percent of females were exposed [in the workplace] an average of 313 minutes and 350 minutes,

respectively." (59 FR 15987) However, close examination of the study itself and a subsequent published report (Ex. 8-168) indicates that the exposure time reported was not actual or continuous but total potential exposure time.[17] The study indicates that while 62 percent of the sample population responded that they were in the presence of smokers at some time during the day, only 27 percent were in work locations with smokers. The Proposed Rule fails to mention these important qualifications.

The Proposed Rule also does not discuss a more recent study of human activity patterns among Californians conducted by the U.S. EPA (1993).[18] Exposures to ETS were assessed in precisely the same way as in the CAPS study, namely, by personal recall diary. The study concludes that "less than 2 percent of the time was spent indoors or in transit in the presence of smokers." The report also notes that personal exposures to particles (daytime averages) did not differ significantly between individuals who reported exposure to ETS and those who did not.

The second population survey mentioned in the Proposed Rule is an undated study, the National Health Interview Study, conducted by the U.S. Centers for Disease Control. (Ex. 8-51) According to the Proposed Rule, the "results suggest that at least 19 percent of employed nonsmokers experience ETS exposure at work." (59 FR 15989) The Proposed Rule characterizes the data in the CDC

study as an "underestimate" because they are "based solely on self-reported information and the question was not very specific in defining immediate work area." (59 FR 15995) This criticism, of course, applies to all the human activity pattern surveys relied upon by OSHA in its analysis of workplace ETS exposures.

A "re-analysis" of a 1986 study is cited as the third human activity pattern survey in the Proposed Rule. (Ex. 8-67) (59 FR 15989, 15995) The original study reported that the workplace was a source of ETS exposure for only 28 percent of all respondents.[19] According to the Proposed Rule, a "re-analysis" of a subset of the data from the Cummings, et al. study purportedly indicates that 49 percent of employed subjects (who report no ETS exposure at home) report ETS exposure at work. (59 FR 15989) OSHA concludes from this that the workplace is a significant source of ETS exposure for nonsmokers. (59 FR 15989) OSHA in fact adopts the 49 percent exposure figure as its upper bound estimate for nonsmoker exposure to ETS in all U.S. workplaces. (59 FR 15995) Again, the applicability of those data -- compiled in 1986 -- to the entire nonsmoking workforce, is not addressed.

The fourth human activity study cited in the Proposed Rule was conducted in 1990 and is offered in support of OSHA's contention regarding "the widespread" exposure to ETS in the workplace. (Ex. 8-98) The survey included 196 nonsmoking

volunteers who provided estimates of their respective exposures to ETS during the work day. According to the Proposed Rule, the study indicates that more nonsmokers are exposed to ETS at work than at home.**** (59 FR 15989) The Proposed Rule does not mention, however, that the volunteers for the study were recruited only from workplaces that permitted smoking. The sample obviously was not randomly selected and therefore cannot serve as the basis of OSHA's claim regarding the magnitude of ETS exposure in the workplace. Interestingly, the study also reports average exposures to ETS of approximately 30 minutes per day. Those data directly contradict OSHA's interpretation of exposure time from the CAPS study (350 minutes of ETS exposure a day). (59 FR 15989)

The four "human activity pattern studies" selected by OSHA do not provide a consistent or complete dataset for estimating the extent of nonsmoker exposure to ETS in the workplace. The data are unverified by actual exposure measures and are not representative of <u>current</u> exposure conditions for ETS in the workplace.

****.A 1994 update of the study reports, contrary to OSHA's claim, that (1) volunteers who lived with smokers had significantly higher cotinine concentrations than those who were exposed to ETS in the workplace; (2) 62 percent of all respondents rated their exposure to ETS outside the home as "low"; and (3) 47 percent of those reporting ETS exposures at work had nondetectable cotinine concentrations. (See: Emmons, K., et al., "An Evaluation of the Relationship between Self-Report and Biochemical Measures of Environmental Tobacco Smoke Exposure," <u>Preventive Medicine</u> 23: 35-39, 1994)

Smoking in the workplace is currently regulated through a combination of private initiatives and government restrictions. The Proposed Rule does not consider the impact of smoking bans and other kinds of smoking restrictions on its estimates of nonsmoker exposures to ETS in the workplace. Although the Proposed Rule cites a survey conducted by the Administrative Management Society Foundation indicating that 25 percent of companies ban smoking, this and other information on smoking restrictions is not factored into OSHA's estimate of current exposure to ETS in the workplace. (59 FR 16017) For example, the Proposed Rule does not discuss a 1991 Bureau of National Affairs survey of 833 companies regarding workplace smoking policies.[20] The survey reports that 85 percent of the companies responding had smoking policies in 1991, up from 54 percent in 1987 and 36 percent in 1986. Thirty-four percent of the surveyed companies had total bans on smoking, compared with just 7 percent in 1987 and 2 percent in 1986. Moreover, data from the survey indicate that smoking was banned in all open work areas in 80 percent of the responding companies in 1991, compared with a ban in open work areas in only 51 percent of companies in 1987. Obviously, the data indicate a severe tightening of restrictions on smoking in the workplace from 1986 through 1991. Other data indicate a continuation of the trends on smoking restrictions through 1994.[21,22,23] Smoking restrictions will of course reduce the percentage and extent of nonsmoker exposure to ETS in the workplace -- a point completely ignored in OSHA's analysis.

The failure to utilize current data on ETS exposures in the workplace invalidates OSHA's conclusions that "the workplace is a major location of ETS exposure to nonsmokers" and that "exposure to ETS is pervasive." (59 FR 15990, 16007) OSHA's analysis of "significant risk" for ETS assumes that the proportion of nonsmoking workers exposed to ETS in the workplace remains constant over time. (59 FR 15996) That assumption is invalid. Changing patterns of smoking, decreased percentages of smokers,***** increased numbers of smoking restrictions and improved ventilation in the workplace will all affect estimates of nonsmoker exposure to ETS.

For an accurate representation of ETS exposure, OSHA must therefore determine (1) how many smokers are employed; (2) how many are permitted to smoke at work and under what conditions; (3) how many worksites are adequately ventilated; (4) and how (1)-(3) will affect actual ambient exposures to ETS. The Proposed Rule does not address any of these conditions.

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*****.For example, a recent analysis by the Centers for Disease Control reports that less than 27 percent of the adult population smokes, down from 33 percent ten years ago. (The Wall Street Journal, May 20, 1994).

The Proposed Rule's discussion of ambient levels of ETS constituents in the workplace is selective and incomplete; the studies cited are outdated and unrepresentative of current exposure levels reported for ETS in the workplace

The Proposed Rule discusses a small and select group of ETS measurement studies from homes and other venues in order to provide an average estimate of ETS concentrations in the workplace. (59 FR 15990-1) The studies cited by OSHA employ nicotine and respirable suspended particles (RSP) as markers for ETS exposures. According to OSHA's analysis, studies in offices report "a range of average nicotine concentrations" of 2 to 10 micrograms per cubic meter (ug/m^3), and a range for RSP exposures of 18 to 95 ug/m^3. (59 FR 15990) The "average" exposure for nicotine is later "adjusted" to "5 to 10 ug/m^3" for the average worker and "50 to 100 ug/m^3" nicotine for the most-exposed individuals. (59 FR 15991)

The Proposed Rule cites no published studies on ambient nicotine or RSP measures in the workplace after 1991. This omission includes two major literature reviews on the issue of ETS markers and measurement.[8,24-29] Other pre-1991 studies on nicotine and RSP measurements in the workplace, involving hundreds of offices and restaurants, are not considered in OSHA's analysis.[30-38] (See Table V) The latter studies, together with studies on other markers for ETS exposures in the workplace, were, however,

TABLE V

NONSMOKER EXPOSURE TO ETS
RECENT WORKPLACE AIR MONITORING STUDIES:
NICOTINE AND RESPIRABLE PARTICLES*

Study	Sample	Nonsmoker Exposure; (Mean; ug/m³)	Comment
Hedge, et al. (1993) USA	27 office buildings Area sampling	2.4 ug/m³ - nicotine 10.2 ug/m³ - RSP/UVPM**, ***	Smoking restricted to offices and open-plan cubicle workstations; ("a nonsmoker may be exposed to the nicotine content of about 3 cigarettes per year.")
" "		3.8 ug/m³ - nicotine 5.8 ug/m³ - RSP/UVPM	Smoking restricted to areas with no local treatment of air; ("[nonsmoker exposure equivalent of] about 5 cigarettes per year.")
Lambert, et al. (1993) USA	7 restaurants	1.0 ug/m³ - nicotine 27.8 ug/m³ - RSP	Median levels for nonsmoking sections.
Broder, et al. (1993) Canada	3 office buildings	23 ug/m³ - RSP 14 ug/m³ - RSP	Before smoking ban. After smoking ban.
Holcomb (1993)	5 studies; 270 samples, offices and public buildings	0.3 ug/m³ - nicotine	Review article.
	24 studies; 640 samples, offices and public buildings	45.9 ug/m³ - RSP	
Turner, et al. (1992) USA	585 offices	0.17 ug/m³ - nicotine 20.11 ug/m³ - RSP	254 nonsmoking offices; ("Spillover from smoking areas to nonsmoking areas appears to be minimal.")
Guerin, et al. (1992)	Review of published nicotine data for offices, restaurants, public buildings and transportation facilities		"Nicotine concentrations are generally at least one and up to three orders of magnitude lower than the eight hour time-weighted permissible exposure limit of 500 ug/m³ specified by OSHA for workplace exposure."
Oldaker, et al. (1992) USA	4 office buildings	2.75 ug/m³ - nicotine 21 ug/m³ - RSP/UVPM	Unrestricted smoking (2 buildings).

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* Studies include 700 offices and 150 restaurants that were not considered in OSHA's proposed rule on ETS.

** RSP = respirable suspended particulate; UVPM = ultra-violet particulate matter (identifies ETS contribution to indoor levels of RSP).

*** OSHA's permissible exposure limit (PEL) for nicotine is 500 ug/m³; the PEL for respirable particulate is 5,000 ug/m³.

Study	Sample	Nonsmoker Exposure; (Mean; ug/m³)	Comment
Oldaker, et al. (1990) USA	125 offices	4.8 ug/m³ - nicotine 27 ug/m³ - RSP/UVPM	Average for both smoking and nonsmoking areas.
" "	82 restaurants	5.1 ug/m³ - nicotine 36 ug/m³ - RSP/UVPM	Average for both smoking and nonsmoking areas. "Estimated mean exposure for an eight-hour day in an office is 0.02 cigarette equivalent and for a 1-L meal in a restaurant, 0.003 cigarette equivalent."
Vaughan and Hammond (1990) USA	Office building 30 sample locations	2.0 ug/m³ - nicotine 0.3 ug/m³ - nicotine	Before smoking restrictions. After smoking restrictions.
Crouse, et al. (1989) USA	42 restaurants	5.9 ug/m³ - nicotine 26.1 ug/m³ - RSP/UVPM	"Average exposure to ETS is 50 to 1,000 times lower than exposure [to] a single cigarette."
Proctor, et al. (1989) UK	Office building 10 samples	0.6 ug/m³ - nicotine 8.8 ug/m³ - RSP/UVPM	Median levels.
Thompson, et al. (1989) USA	35 restaurants 6 food courts Personal nicotine monitor	5.4 ug/m³ - nicotine 2.3 ug/m³ - nicotine	Overall exposure.
Carson and Erikson (1988) Canada	31 offices	7.2 ug/m³ - nicotine**** 24 ug/m³ - RSP/UVPM	"Exposure estimated from mean nicotine and UV-PM results were 0.004 and 0.001 cigarette equivalent per hour, respectively."
Sterling and Mueller (1988) Canada	Office building 8 nonsmoking locations receiving recirculated air from a designated smoking area	1.0 ug/m³ - nicotine 8 ug/m³ - RSP	"Equivalent to 1/1800 of the nicotine [per hour] inhaled by actively smoking one cigarette."

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**** Incorrectly cited in the OSHA NPR.

submitted to the docket of the OSHA RFI on Indoor Air in 1992. (Ex. 3-1074)

The studies on ambient measures of nicotine in offices and restaurants that were omitted from OSHA's analysis report average levels of approximately 2.0 and 3.5 ug/m^3 nicotine, respectively. These exposures are equivalent to 1/400 to 1/200 of the nicotine found in a single cigarette. Averages for nonsmoking areas in workplaces with smoking restrictions are even lower, averaging less than 1 ug/m^3 nicotine, or about 1/1,000 of the nicotine in a single cigarette. This means that the typical nonsmoking worker would have to spend from 200 to more than 1,000 hours in an office, restaurant or public place in order to be exposed to the nicotine equivalent of a single cigarette.[26,31,32,35-36] Moreover, these exposures are 100 times lower than OSHA's PEL for nicotine, 0.5 mg/m^3. (29 CFR 1910.1000, Table Z-1)

Respirable suspended particles (RSP) are contributed by a number of sources, a point acknowledged in the Proposed Rule. (59 FR 16003) OSHA states:

> People contribute millions of particles to the indoor air primarily through the shedding of skin scales. Many of these scales carry microbes, most of which are short-lived and harmless. Clothing, furnishings, draperies, carpets, etc. contribute fibers and other fragments. Cleaning processes, sweeping, vacuuming, dusting normally remove the larger

particles, but often increase the airborne concentrations of the smaller particles. Cooking, broiling, grilling, gas and oil burning, smoking, coal and wood generate vast numbers of airborne indoor pollutants in various classifications.

Results of a 1993 study on particle exposures conducted by the U.S. EPA indicate that one hour of cooking activity generates approximately 100 times the particle concentration from a single cigarette.[39] The study reported that average percent contributions from "fine particle mass" (RSP) were: 10 percent from smoking, 68 percent from outdoor sources and 22 percent from unidentified sources.[39]

Special methods have been developed for the analysis of airborne particles that determine the relative contribution of ETS to total particles in the indoor air. One analytic technique employs ultraviolet particulate matter (UV-PM) as an indicator of the particle fraction contributed to the air by ETS.[8] Studies of UV-PM measurements in offices report ranges of from 27 to 44 ug/m^3 in areas where smoking is permitted.[24] Although indoor exposure limits do not exist for ETS-related particles, the World Health Organization (1992) has designated 100 ug/m^3 or less as an exposure level of "limited or no concern" for particles contributed by ETS.[40]

A recent review has summarized average levels of airborne particles from smoking and nonsmoking areas reported in studies

performed in various settings.[24] The average particle concentration in the air of homes with smokers is about 27 micrograms per cubic meter higher than in homes of nonsmokers; offices and public places that permit smoking report average levels of particles that are about 22 micrograms/cubic meter higher than those reported for nonsmoking locations, and smoking areas of restaurants report levels that are 42 micrograms/cubic meter higher, on average, than nonsmoking areas.

In the largest single study of its kind, researchers monitored 585 offices for various ETS-related constituents, including particles.[29] The average level of particles recorded for 331 offices in which smoking was permitted was 46 ug/m^3, compared with an average of 20 ug/m^3 for 254 nonsmoking offices. These levels are all far below that designated by the WHO as "limited or no concern."[40]

The Proposed Rule contends that the contribution of RSP by smoking is much greater than that reported in studies employing the UV-PM method. The Proposed Rule combines the results of three studies of smoking and nonsmoking buildings conducted in the early 1980s. The combination of data from the three studies provides an average estimate of 262 ug/m^3 RSP in smoking buildings, compared with 36 ug/m^3 in nonsmoking buildings. (59 FR 15990) An analysis identical to that presented in the Proposed Rule occurs in a 1992

paper written by two antismoking activists.[41] (The paper, however, is not referenced in the Proposed Rule.) Careful examination of the three studies cited reveals that the data therein do not support the contention of the Proposed Rule. One of the studies measured RSP levels in pizzerias, bars, lodge halls, bowling alleys, church bingo games and fast food restaurants in 1980. The only office measurement reported was conducted under experimental conditions in a room in which levels of ETS were generated by seven smokers smoking 32 cigarettes in 49 minutes, yielding RSP values as high as 500 micrograms per cubic meter. One of the other studies cited included no office measurements. Such selective combinations of data only serve to confuse the issue. The studies clearly do not represent typical workplaces at 1994 levels of ETS exposure.

According to a comprehensive review undertaken by researchers at Oak Ridge National Laboratories, "chemical means for estimating the contribution of ETS to RSP have been evaluated, and suggest that ETS-RSP may comprise from 10 percent to 50 percent of indoor air RSP, in the field scenarios to which the methods have been applied."[8] Indeed, even a cursory examination of actual studies, consisting of hundreds of office, restaurant and other workplace situations, reveals that source-apportioned RSP levels due to ETS are at levels 5 to 10 times lower than those suggested in OSHA's Proposed Rule.[24, 29, 31-33]

ETS constituent levels in the workplace: an interpretation

With the exception of nicotine, solanesol and 3-ethenylpyridine, none of the substances used as markers for ETS in the air is characteristic of tobacco smoke. Other sources such as heaters, stoves, building materials, cleaning products and human activities often generate greater levels of those substances than ETS.[8] For example, studies indicate that automobile combustion, industrial processes, home heating and gas cooking are the predominant sources of carbon monoxide and nitrogen oxides in indoor air.[24] For this reason, neither constituent is an appropriate marker for ETS. Researchers report that there is little difference in ambient levels of carbon monoxide or nitrogen oxides in smoking and nonsmoking areas of workplaces and public places and in homes with or without smokers.[8,24,42-46] Levels of volatile organic compounds such as formaldehyde and benzene in the presence of smoking are often indistinguishable from levels reported in nonsmoking areas.[8,32,47-51] Studies that have examined ETS constituent levels of nitrosamines[52-54] also report minimal contributions to overall ambient air levels in homes, offices and public places.

Nicotine and RSP were selected by OSHA as the most suitable markers for assessing exposure to ETS. While nicotine is characteristic of tobacco smoke in the ambient air, it represents

a gas-phase constituent and indicates nothing about the particle (RSP) phase of environmental tobacco smoke exposures. RSP, on the other hand, is not characteristic of tobacco smoke, but, if correctly sampled and analyzed, can provide approximations of exposure to the particle phase of ETS. Measurements of both nicotine and RSP are typically reported in terms of micrograms per cubic meter (ug/m^3). The absolute values of the levels measured, however, may not be meaningful to anyone other than scientifically trained personnel. It is therefore convenient to refer to a comparative or illustrative device in order to facilitate understanding about the levels of exposure to nicotine and RSP as reported in the scientific literature.

One particularly useful illustrative means for representing reported levels of nicotine and RSP in the ambient air is the notion of "cigarette equivalents." The use of "cigarette equivalents" as a heuristic device for understanding ETS exposures does not imply that active smoking and nonsmoker exposure to ETS are the same thing, or that mainstream smoke is "equivalent" to ETS. Nor does it mean that a measure of nicotine or RSP in terms of "cigarette equivalents" represents exposure to other constituents in ETS. However, the concept of "cigarette equivalents" offers a convenient means for quantitative comparisons of ETS exposures, and is a commonly used method for reporting measured levels in the published literature.[24, 26, 31, 32, 36, 55]

For example, Oldaker, et al. reported average levels of nicotine exposure of 4.8 ug/m^3 for 125 offices.[31] The authors estimated exposures to nicotine for an eight-hour work day in an office to be equivalent to 0.02 of a cigarette. The authors also measured nicotine levels in 82 restaurants and the resulting average concentration was 5.1 ug/m^3; the exposure equivalent for a one-hour meal was 0.003 of a cigarette.

Carson and Erikson, cited in OSHA's Proposed Rule (59 FR 15991), measured nicotine and RSP (UV-PM) in 31 offices.[55] The authors wrote: "Exposure estimated from mean nicotine and UV-PM results were 0.004 and 0.001 cigarette equivalent per hour, respectively." Proctor measured nicotine levels in smokers offices and reported that the average level of 3.1 ug/m^3 nicotine would provide an exposure equivalent of 0.018 to 0.010 of a cigarette over the course of a work-day.[32] Proctor writes: "This means that a male nonsmoker would have to work in the smoker's office for over 11 weeks before being exposed to the equivalent nicotine as from smoking one cigarette." UV-PM/RSP levels in smokers' offices were reported at 24 ug/m^3.[32] This would translate into an exposure equivalent of 0.013 cigarettes per day. Thus, Proctor writes: "This again would result in a male nonsmoker working in the smoker's office for 15 weeks before being exposed to the equivalent particulates as smoking one cigarette."

Hinds and First, in one of the first studies of its kind, measured nicotine levels in various public places in 1975.[36] The venues included waiting rooms, restaurants, and cocktail lounges. Measured levels of nicotine ranged from 1.0 to 10.3 ug/m^3, translating into equivalents of 0.001 to 0.009 cigarette per hour.

In 1993, Hedge, et al. measured nicotine in 27 air-conditioned offices under various smoking policies.[26] The authors write: "[A] nonsmoking office worker in open offices who does not spend time in designated smoking areas on average may be exposed to the nicotine content of about three cigarettes per year."

Although a suitable internal dose marker for ETS is not available, scientists have estimated that ETS-related particle uptake in the typical nonsmoker is approximately two-hundredths of one percent (0.02 percent) that of the active smoker.[56-58] Other scientists have estimated that ETS "inhaled doses" (based on particles) are 10,000 to 100,000-fold less than average doses calculated for active smokers.[59] The estimated nonsmoker "dose" is equivalent to actively smoking less than one cigarette over the course of a year.

Another scientist recently calculated estimated "retained doses" for nonsmokers using average exposures to various ETS constituents reported in the published literature.[24] The calculated

dose for tobacco smoke particles, expressed in terms of cigarette equivalents, ranges from less than one-half to about four cigarettes per year for "light" and "heavily-exposed" nonsmokers, respectively.

The Proposed Rule does not utilize actual ambient exposure measures for ETS in the workplace in its determination of "significant risk"; exposure estimates from epidemiologic studies on ETS are used in the determination of "significant risk"; exposure estimates from epidemiologic studies are not quantifiable; they are unreliable and inconsistent with actual ambient measurements of ETS

In its analysis of "significant risk" for ETS, OSHA employs two epidemiologic studies on lung cancer and heart disease in nonsmokers. (Exs. 8-106, 8-139) (59 FR 15995). Exposures to ETS were determined in those studies through the use of questionnaires that, in turn, were dependent upon the accuracy of recall of exposure among those questioned. No actual ambient exposure measures were included in the epidemiologic studies. **OSHA's entire discussion of ambient measures of ETS exposures in the workplace is omitted from its significant risk analysis and replaced by the exposure estimates from two epidemiologic studies on nonsmoker lung cancer and heart disease.**

One critical source of bias in epidemiologic studies on ETS is called "exposure misclassification bias" and arises from

errors in reporting ETS exposures.[5,60-65] As was suggested earlier, epidemiologic studies do not measure actual ETS exposures, but rather employ questionnaires to generate estimates of ETS exposure over long periods of time. Sometimes, the studies use exposure estimates given by spouses, children, or next-of-kin. The questionnaires used in epidemiologic studies on ETS vary and have not been validated or checked for accuracy. The National Research Council and other authors have criticized the use of questionnaires as representations of accurate and comprehensive exposure histories in ETS studies.[66,67] Other studies demonstrate that questionnaires are an unreliable and inaccurate measure of ETS exposure.[14-16,68-69]

Typically, questions regarding ETS exposures in epidemiologic studies are of the following sort: "Are you married to a smoker?"; "How long have you lived with a smoker?"; "About how many cigarettes a day did the smoker smoke in your presence?"; "About how many cigarettes a day did the smoker smoke?"; etc. Answers to such questions vary widely across studies. For example, Friedman, et al. noted that, in their study, 30 to 35 percent of nonsmokers who were married to smokers reported no exposure whatsoever to ETS.[15] A more recent study reported a 36 percent exposure misclassification rate when ETS exposures determined by questionnaires were compared to exposures determined by ambient air measurements.[16] The authors of that study conclude: "The results . . . suggest that questionnaires lead to a large amount of

misclassification which must be taken into account when assessing the effect of ETS exposure" and that "an objective method to measure exposure which is sensitive, accurate, and reliable is needed to validate them." Another recent study that compared ETS measurements with questionnaire responses found that "the false report rate was high. Among those 575 participants reporting an average of 42 or more hours/week [exposure to ETS], 58 percent did not have a detectable cotinine level."[68]

Coultas, et al., in a study cited in the Proposed Rule (Ex. 8-66), report that "the levels of cotinine, respirable particles, and nicotine varied widely with self-reports of exposure to ETS."[12] A second study by Coultas, et al. reports that "personal exposure also varies with the nonsmoker's proximity to the smoker. Questionnaires cannot comprehensively and accurately assess each of these factors. Not surprisingly, we found that the questionnaire responses were poor predictors of concentrations of respirable particles and nicotine."[13] Schenker, et al., in another exposure assessment study, concluded that "self-reported exposure to ETS is an inaccurate measure of passive smoking in the occupational setting."[63]

Pron, et al. noted in their study that "responses to initial screening questions used to detect a person's exposure to passive smoke were more reliable for residential than for

occupational exposure . . . [Q]uantitative measures of exposure to passive smoke, i.e., number and duration of exposure, were even less reliably reported."[62] In another study by Coultas, et al., the authors reported that "responses concerning recent tobacco smoke exposure and urinary cotinine levels were correlated only to a modest degree." The authors "conclude that adults can reliably report whether household members smoked during their childhood, but information on quantitative aspects of smoking is reported less reliably."[69] Lerchen and Samet in 1986 reported that spouses of smokers correctly reported the smoking status of their spouses, however, "for the number of cigarettes smoked per day, wives tended to report 20 cigarettes smoked even when their husband smokes substantially more or less."[64]

OSHA argues, within the context of its analysis of "significant risk" for ETS, that home "exposures" to ETS are comparable to workplace exposures. It is argued that "in the absence of purely occupational data, information derived in environments other than worksites is also considered." (59 FR 15994) The Proposed Rule cites a report prepared by Meridian Research in 1988 that contends that "it is the exposure to environmental tobacco smoke, and not the environment in which that exposure occurs, that is the important risk factor." (Ex. 8-221) The Proposed Rule continues: "Therefore, health effects observed and the risk estimates calculated from studies of the general

population, or of selected subgroups, such as nonsmoking wives of smoking husbands, are relevant to the working nonsmoking population." (59 FR 15994)

There are at least three distinct issues regarding OSHA's argument. First, OSHA equivocates on the phrase "exposure data." OSHA proposes to replace actual ambient air measures of ETS constituents in the workplace with exposures assessed by questionnaire response in epidemiologic studies. The foregoing analysis clearly indicates that questionnaire responses cannot quantitatively approximate, much less replace, actual measures of ETS constituents in the air of workplaces.

The second issue raised in OSHA's argument concerns the comparability of actual levels of ETS constituents in the home and in the workplace. OSHA contends (but does not prove) that the levels are comparable. (59 FR 15994) Recent data, however, indicate that this is not the case. In a 1993 study of 96 nonsmoking, married women, ETS exposures were continuously monitored over a one-week period.[70] The results for actual measures of ETS exposures (using 3-ethenylpyridine and nicotine as markers for ETS) indicate that workplace exposure is "tenfold lower than home exposure due to living with a smoker." Average values for exposure to nicotine in the workplace indicate that it is a trivial source of (ETS) exposure (median value, 0.21 ug/m^3 nicotine,

compared to the nicotine delivery of a single cigarette: 880 ug/m^3).[70]

The third issue raised in OSHA's argument is whether or not data from household "exposures" to ETS can be applied to the workplace. If current published data regarding workplace exposure to ETS constituents were not available, then perhaps OSHA could make its argument. However, current workplace exposure data are available, including seven major studies and two major review surveys of ETS measurements in workplaces, that could have been used by OSHA in its analysis. It is clear that OSHA chose not to use the data from those studies and reviews because, in most instances, the data reveal that ETS exposures in the workplace are minimal and often indistinguishable from background levels. Even in the complete absence of appropriate data on ETS measurements in the workplace, OSHA would still have to justify its argument for the application of exposure data from epidemiologic studies in the home to the workplace. As one scientist notes:

> Because workers not exposed to ETS and workers exposed to ETS in the same occupational group have not been medically followed for several years, there is no justification to apply the reasoning that because home exposure through spousal smoking might be associated with cardiopulmonary disease, then workplace ETS exposure can be expected to have a similar association . . . . Results of household exposure cannot be applied to workers . . . . Confounding factors related to ETS exposure in workplaces are different from factors

influencing spouses and children in studies of household exposure. Dietary factors and household pollution may influence the incidents of spousal and childhood diseases. On the other hand, workers are exposed to industrial chemicals and outdoor pollutants as well as work-related stress. Familial patterns of inherited or acquired susceptibility to cardiopulmonary disease do not apply to occupational groups . . . . Unlike household exposure, workplace exposure can be monitored by good industrial hygiene practice.[2]

Recent data on ETS exposures in the workplace based upon personal monitoring of ETS constituents undermine OSHA's assumptions regarding the extent and frequency of ETS exposures, the comparability of home and workplace ETS exposure levels, the ineffectiveness of dilution ventilation for the minimization of ETS exposures and the accuracy of questionnaires and biomarkers for the quantitation of ETS exposures

The results from three recent independent studies on personal exposures to ETS in the workplace are now publicly available. The first study, conducted by indoor air quality specialists from British Columbia, Canada, assessed personal exposures to ETS constituents among 25 nonsmoking subjects in two office buildings in Richmond, Virginia.[71] The personal monitors recorded levels of exposure to four indicators of particle-phase ETS and two constituents of vapor-phase ETS, including nicotine. Smoking was unrestricted throughout the buildings and the

ventilation systems were operated in accordance with the outdoor air ventilation rates specified in ASHRAE Standard 62-1989. Approximately 20 percent of the total workforce in the two buildings were active smokers.

Average exposures to total respirable suspended particulate ranged from 23 to 29 ug/m^3; the average range for nicotine concentrations was 2.04 to 2.71 ug/m^3. Correlations among cotinine levels, questionnaire responses regarding the frequency of smoking and measured levels of particle and vapor-phase indicators were very weak, suggesting that both questionnaire responses and the use of cotinine are poor surrogates for quantitating exposures to ETS constituents. Measured exposure levels of particle and vapor-phase constituents in this study were two to three times lower than levels estimated in OSHA's Proposed Rule.

Researchers from Oak Ridge National Laboratories reported preliminary results from an on-going study of personal exposures to ETS in May, 1994.[72] Participants in the study wore personal sampling pumps that collected various ETS particle and vapor-phase constituents. The samplers were operated during work hours and in all locations away from work. Preliminary results indicate that the range of average respirable suspended particulate levels in the workplace was from 10 to 30 ug/m^3; average nicotine levels ranged between 0.05 and 0.1 ug/m^3. Median time-averaged exposure levels

for respirable suspended particulate and nicotine in a smoking workplace were 20.8 ug/m^3 and 0.243 ug/m^3, respectively. For the nonsmoking workplace, average RSP exposure levels were 15.1 ug/m^3 and average nicotine levels were 0.034 ug/m^3.

Time-averaged exposure levels of nicotine in a smoking home were 4.35 times higher than average levels reported for workplaces in which smoking was permitted (0.827 vs. 0.190 ug/m^3).[72] Measurements of ETS-related particles (UVPM, FPM, and solanesol) indicated nearly three times the exposure in a smoking home compared to a smoking workplace, even though all the absolute values of the ETS-related particles were low. Average salivary cotinine levels did not effectively separate individuals who lived in a nonsmoking home and who worked in a smoking workplace, and individuals from a nonsmoking home and nonsmoking workplace (0.36 vs. 0.11 ng/mL).

A similar study conducted in the United Kingdom by researchers from Hazelton Laboratories was completed in 1993. Two hundred and fifty-five participants were monitored for personal exposures to ETS vapor-phase and particle-phase constituents.[73] Average levels of ETS constituent exposure were low -- over five times lower than the levels estimated in OSHA's Proposed Rule (e.g., seventy percent of the subjects were exposed to less than 10

ug/m^3 of ETS-related particles and over 60 percent were exposed to less than 1 ug/m^3 of nicotine).

The Hazelton researchers also ranked relative contributions of ETS constituents from work, home, travel and leisure locations. Data gathered through questionnaires about relative contributions of ETS from each venue were compared with actual measures of ETS constituents. The results indicate that subjective estimates of exposure tended to rank as higher the relative contributions of ETS from leisure, work, home and travel, respectively. Direct ambient air measurements indicated that the ranking of relative contributions to total ETS exposure was: the home, leisure venues, work and travel. Subjective assessments appeared to overestimate contributions of ETS from both workplace and leisure venues.

Mean exposure levels for various ETS constituents were greater for subjects with a smoking spouse or partner than for those with a nonsmoking spouse or partner, but the distribution of the results did not provide a clear distinction between the two groups. Forty-six percent of subjects with a smoking spouse or partner assessed their ETS exposure as "none" or "low." This assessment was supported by direct ambient measurements. Approximately 30 percent of subjects with a smoking spouse or

partner assessed leisure or work as their principal source of exposure.

The final objective of the study was to compare questionnaire responses, direct ambient air measurements and salivary cotinine levels as methods for assessing exposure to ETS. Direct measurements by personal monitoring appeared to provide the most reliable overall estimates of ETS exposures. The data indicate that salivary cotinine levels correlated very poorly with direct ambient air measurements of ETS constituents. Some subjects who had been exposed to high levels of ETS-related particulate and nicotine had no detectable salivary cotinine levels, and some subjects who had not been exposed to any measurable quantity of ambient nicotine had relatively high levels of salivary cotinine.

The TDSA, Oak Ridge and Hazelton studies demonstrate the viability and accuracy of personal monitoring for ETS constituents. The studies reaffirm the inaccuracies inherent in the use of questionnaires and cotinine for the quantitative assessment of ETS exposures. The overall results indicate relatively low contributions of ETS-related constituents to total pollutant burdens for individuals exposed to tobacco smoke in the workplace. The data tend to undermine the claims that ETS exposures are "ubiquitous" and that levels of ETS encountered at work are comparable to levels encountered in other venues.

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SECTION V

MATERIAL IMPAIRMENT: LUNG CANCER

LUNG CANCER

OSHA'S PROPOSED RULE FAILS TO DEMONSTRATE THAT CURRENT ETS EXPOSURES IN THE WORKPLACE POSE A SIGNIFICANT RISK OF MATERIAL HEALTH IMPAIRMENT DUE TO LUNG CANCER IN EXPOSED WORKERS

At Section II.C.6.(a), OSHA's Proposed Rule claims that the "results of epidemiological and experimental studies indicate that exposure to ETS is causally associated with cancer of the lung in chronically-exposed nonsmokers." (59 FR 15979) At Sections IV.A. through IV.D. (59 FR 15992-15996), OSHA then proceeds to conduct a "Preliminary Quantitative Risk Assessment" for lung cancer.

However, OSHA's Proposed Rule fails to provide adequate support for the claim of a causal association, or to validate the decision to conduct a risk assessment. OSHA focuses inappropriately on data on "spousal smoking," rather than considering in full the available epidemiologic data on ETS exposures in the workplace. Moreover, OSHA's review of the scientific literature relevant to this issue is incomplete and inaccurate, as presented in the text, sometimes including misrepresentations of the results and conclusions of certain studies.

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Due to its failure to focus on the best available data on workplace ETS exposures and to its numerous errors and omissions, OSHA's Proposed Rule does not adequately demonstrate a significant risk of material health impairment due to lung cancer in persons exposed to ETS in the workplace.

OSHA FAILS ADEQUATELY TO SUPPORT THE POSITION THAT ETS EXPOSURE IN THE WORKPLACE IS CAUSALLY ASSOCIATED WITH LUNG CANCER IN NONSMOKERS

At Section II.C.6.(a) (59 FR 15979), OSHA claims that data from epidemiologic studies, "taken in the aggregate," support a causal association between ETS exposure and nonsmoker lung cancer. OSHA does not provide references in support of this claim.

In subsequent text, OSHA references the 1992 EPA Risk Assessment on ETS (Ex. 8-311) for that report's discussion of active smoking studies. OSHA invokes the argument of "biological plausibility," suggesting that unspecified qualitative similarities between mainstream smoke and ETS support an analysis of cancer risk due to exposure to the latter. This argument does not withstand critical analysis, such as recognition of the distinct chemical differences among mainstream smoke, sidestream smoke, and ETS detailed elsewhere in this submission. OSHA's failure to

differentiate among different forms of tobacco smoke pervades this section of the Proposed Rule.

The EPA Risk Assessment on ETS suggested that, because mainstream smoke reportedly contains over 40 "known" or "suspect" carcinogens, so must ETS. (Ex. 8-311) However, the EPA's claim is not convincingly supported by scientific data. The EPA concedes in its report that specific substances responsible for the supposed carcinogenicity of mainstream smoke have never been identified, and that associations between "suspect" carcinogens in mainstream smoke and specific diseases have never been established.

Moreover, most of the claimed "carcinogens" in mainstream smoke have never been tested via inhalation in animals (nickel and polonium-210 have reportedly produced human-type lung tumors in animal studies, but these substances are derived from a number of sources).[1,2,3]

To further support its claim of biologic plausibility, OSHA references a study by the Centers for Disease Control, which reported that cotinine, a metabolite of nicotine, was detected in samples of bodily fluids of all the persons surveyed in the study. (Ex. 8-50) OSHA interprets these reported results as "indicating that everyone in the sample had detectable exposure to tobacco smoke." (59 FR 15980) It is an oversimplification to assume that

any detectable level of cotinine in bodily fluids necessarily implies exposure to tobacco smoke. Nicotine is also present in several foods (potatoes, tomatoes, eggplant, tea); detectable cotinine levels may be produced following the ingestion of reasonable amounts of these foods.[4,5,6] At the sensitive levels of detection employed in the CDC paper (to 0.030 ng/mL), cotinine related to dietary nicotine could certainly be detected. Therefore, any extrapolation of these reported results to the classification of persons as "exposed" to ETS should take diet into account, rather than immediately presuming that detectable cotinine must be due to ETS exposure.

OSHA'S RELIANCE ON DATA ON "SPOUSAL SMOKING" IS INAPPROPRIATE; OSHA FAILS TO DISCUSS RELEVANT AVAILABLE DATA ON REPORTED WORKPLACE ETS EXPOSURES IN THE WORKPLACE

In Section II.C.6.(b), OSHA states that there are "at least 32" epidemiologic studies relevant to a discussion of ETS and lung cancer. OSHA fails to note that the approximately 35 studies publicly available primarily deal with household exposure, assessed most commonly as "spousal smoking." Fourteen studies provide data on reported workplace exposures, which are directly relevant to OSHA's jurisdiction; however, OSHA fails to discuss the workplace data in their entirety.

Taken as a whole, the data on ETS exposures in the workplace do not support a conclusion of increased lung cancer risk

As OSHA's jurisdiction is the workplace, not the home, the spousal smoking data are not the best available data for OSHA to use in promulgating its Proposed Rule. While OSHA erroneously characterizes the spousal smoking studies as studies of "nonsmoking housewives" (59 FR 15993), not all the women included in the studies were "housewives"; in fact, a number of the studies cited by OSHA report estimates of lung cancer risk for workplace ETS exposure. (Exs. 8-36, 8-47, 8-106, 8-119, 8-164, 8-171, 8-192, 8-283, 8-292, 8-326) Additional workplace data are found in studies not referenced by OSHA.[7-9]

The availability of risk estimates for workplace exposure was pointed out to OSHA in Philip Morris' submission to Docket H-122 in response to OSHA's Request for Information on Indoor Air (Ex. 3-1074, response to question 2(a)iii), and in a number of other submissions to Docket H-122. (e.g., Exs. 3-331, 3-1067, 3-1073) While those submissions present detailed discussions of the workplace data, a restatement of the available workplace data is nevertheless presented here. This discussion also includes studies that have appeared since the RFI docket closed.

Of the 14 epidemiologic studies on spousal smoking which include an estimate of workplace ETS exposure, nine were performed in the United States, two in European countries and three in Asian countries. The following are brief descriptions of these studies (in alphabetical order) and their conclusions regarding workplace ETS exposure. Relevant information about the studies (sample sizes, gender of study participants, definition of exposure, the reported point (risk) estimates for workplace ETS exposure, and the statistical significance of the point estimates) are summarized in Table 1.

In a study published in 1992, Brownson and colleagues reported on results of a case-control study of Missouri women who were lifetime nonsmokers or former smokers. (Ex. 8-36) This study is notable for its large sample size, as over 600 lung cancer cases were enrolled, more than 400 of whom were self-reported lifetime nonsmokers. The authors wrote:

> In general, there was no elevated lung cancer risk associated with passive smoke exposure in the workplace (not shown in table). Only lifetime nonsmokers showed a slight increase in risk at the highest quartile of workplace exposure (OR = 1.2; 95% CI = 0.9, 1.7).

As the above paragraph indicates, these authors failed to present their overall risk estimate for workplace smoking, which was apparently not statistically significant. The risk estimate that

TABLE 1: ESTIMATES OF WORKPLACE ETS EXPOSURE AND LUNG CANCER RISK IN NONSMOKERS

STUDY	COUNTRY	NUMBER OF CASES/CONTROLS IN WORKPLACE ANALYSIS	GENDER	EXPOSURE DEFINITION	RISK ESTIMATES
Brownson, et al., 1992	USA	not presented (432/1402 total)	female	"highest quartile" of workplace exposure exposure in the workplace	1.2 (0.9-1.7) "no elevated lung cancer risk"
Butler, 1988	USA	6 cases 7 cases	female male	worked with smoker for 11+ yr worked with smoker for 11+ yr	1.47 (0.15-14.06) 1.72 (0.33-9.04)
Fontham, et al., 1994	USA	609/1247	female	ever exposed	1.39 (1.11-1.74) *
Garfinkel, et al., 1985	USA	14/52 34/118	female	exposure in last 5 years exposure in last 25 years	0.88 (0.66-1.18) 0.93 (0.73-1.18)
Janerich, et al., 1990	USA	not presented (191/191 total)	both	150 person-years exposure	0.91 (0.80-1.04)
Kabat & Wynder, 1984	USA	53/53 25/25	female male	current regular exposure current regular exposure	0.68 (0.32-1.47)[1,2] 3.27 (1.01-10.6)[1,2] *
Kabat, 1990	USA	44/111 37/105	female male	ever exposed at work ever exposed at work	1.00 (0.49-2.06) 0.98 (0.46-2.10)
Kalandidi, et al., 1990	Greece	65/78 (est.)	female	"between extreme quartiles" of exposure some v. minimal exposure exposed at work	1.08 (0.24-4.87) 1.70 (0.69-4.18)[2] 1.39 (0.76-2.54)[1]
Koo, et al., 1984	Hong Kong	2/4	female	exposed at workplace	0.91 (not given)
Lee, et al., 1986	Great Britain	15/158 10/59	female male	ever exposed ever exposed	0.63 (0.17-2.33)[1,2] 1.61 (0.39-6.60)[1,2]
Shimizu, et al., 1988	Japan	not presented (90/163 total)	female	someone at workplace smokes	1.2 (not given) 1.2 (0.70-2.04)[2] 1.2 (0.69-2.01)[1]
Stockwell, et al., 1992	USA	not presented (210/301 total)	female	exposure at work	"no statistically significant increase in risk"

Wu, et al., 1985	USA	not presented (29/62 total)	female	exposed at work	1.3 (0.5-3.3)
Wu-Williams, et al., 1990	China	415/602	female	exposed at work	1.1 (0.9-1.6) 1.22 (0.95-1.57)[2] 1.1 (0.86-1.41)[1]

* statistically significant

1. LeVois, M.E., and Layard, M.W., "Controversy Over Regulating Indoor Air Quality: Environmental Tobacco Smoke," comment submitted to U.S. Occupational Safety and Health Administration, Docket H-122, No. 3-1067, March 19, 1992.

2. Lee, P.N., Environmental Tobacco Smoke and Mortality. Basel, Karger, 1992.

was presented, that is, the one for the most extreme self-reported exposure category, was also not statistically significant.

Another U.S. study is the 1988 Ph.D. dissertation of Butler. (Ex. 8-47) This cohort study followed two groups of California Seventh-Day Adventists, members of a religious sect who adhere to certain lifestyle choices, e.g., abstinence from smoking, caffeine and red meat. Despite the lifestyle differences suggested by membership in this sect, Butler's data have been included in discussions of workplace ETS exposure. For males, Butler reported a risk estimate of 1.72 (95% CI 0.33-9.04); for females, the reported risk estimate was 1.47 (95% CI 0.15-14.06), for having worked with a smoker for eleven or more years. Neither was statistically significant. Despite the large number of individuals enrolled in the study, very few lung cancer cases were obtained. As a result, an extremely small sample size is a major flaw in this study.

Another American study was first published in 1991 by Fontham and colleagues. (Ex. 8-106) It represented a preliminary report on a case-control study of women in five U.S. cities, and is extensively relied upon in the OSHA Notice. For all lung cancer cell types combined, an odds ratio of 1.34 (95% CI 1.03-1.73) was reported for those women who reported that they were ever exposed to ETS in an occupational setting. Fontham and colleagues (1994)

have recently published a report on all five years of the study.[11] They present a "crude" risk estimate for workplace exposure of 1.12 (95% CI 0.91-1.36), which changes dramatically after adjustment for a number of variables to 1.39 (95% CI 1.11-1.74). The magnitude of the change following adjustment is much more pronounced for the workplace risk estimate than for the other risk estimates in the paper. Moreover, the upward increase after adjustment is suspicious. Fontham and colleagues do not address this apparent discrepancy. Because of OSHA's reliance on the 1991 Fontham, et al., study, it is presumed that the second study will be used by OSHA as the rulemaking proceeds. Therefore, a more detailed discussion of the Fontham study will be found in the discussion of OSHA's "risk assessment" in this submission.

Another U.S. study, by Garfinkel and colleagues, was a case-control study of hospitalized women in New Jersey and Ohio, published in 1985. (Ex. 8-119) It included the following estimation of workplace exposure to ETS:

> The interviewer also asked about the average number of hours a day the woman had been exposed to the smoke of others at any time during the past five years, during the past 25 years at home, while at work . . .

The authors reported the following:

> [The] OR for exposure at work during the last five years was 0.88 [95% CI 0.66-1.18; 14 cases, 52 controls]; for the last 25 years, it was 0.93 [95% CI 0.73-1.18; 34 cases, 118 controls].

These two point estimates represent negative associations between reported ETS exposures in the workplace and lung cancer in nonsmokers.

Conclusions based on a case-control study of 191 men and women in New York State were reported in 1990 by Janerich and colleagues (Ex. 8-164), who wrote:

> Exposure in the workplace was measured by recording the number of smokers who worked with each study subject during his or her lifetime and the amount of time the subjects spent working with these smokers. These exposures were compared for case patients and control subjects. Estimating the odds ratio as a continuous variable for an equivalent differential of 150 person-years of exposure gave an odds ratio of 0.91 (95 percent confidence interval, 0.80 to 1.04), indicating no evidence of adverse effect of environmental tobacco smoke in the workplace. [emphasis added]

In 1984, Kabat and Wynder published results of a case-control study of hospitalized individuals, mostly from New York City. (Ex. 8-171) A total of 25 male cases and 53 female cases and their matched controls were included. The authors wrote:

> The plausibility of a role of passive inhalation in lung cancer can be questioned on several grounds . . .
>
> Cases do not differ from controls except for the greater exposure to cigarette smoke at work reported by male cases compared to male controls.

The authors reported that 18 of 25 male cases versus 11 of 25 controls reported being exposed to ETS in the workplace. This result was statistically significant at P = 0.05. The point estimate for workplace exposure of males was reported elsewhere as 3.27 (95% CI 1.01-10.6). However, the authors' reported results for women are inconsistent with their conclusions on men; 26 of 53 female cases versus 31 of 53 controls (i.e., fewer cases than controls) reported exposure in the workplace. This result, a point estimate of 0.68 (95% CI 0.32-1.47), is not statistically significant.

In 1990, Kabat reported preliminary results from an American Health Foundation case-control study, which then included 90 cases and 247 controls.[7] Kabat reported that "preliminary analyses of the data do not indicate any striking ETS exposure differences between cases and controls." Specifically, he reported odds ratios for men of 0.98 (95% CI 0.46-2.10) and for women of 1.00 (0.49-2.06), for reported workplace exposure. The risk

estimate for males is negative, and that for females is the same as the baseline, "no-risk" level.

One of the European case-control studies to assess workplace exposure was conducted on hospitalized women in Athens, Greece.[8] Based on 89 cases and 118 controls, the authors conclude: "The effect of exposure to passive smoking at work was very small and not statistically significant (the RR between extreme quartiles was 1.08 (0.24-4.87))."

Koo and colleagues, in their 1987 case-control study of women in Hong Kong, included an assessment of workplace exposure in an accounting of total lifetime exposure to ETS. (Ex. 8-183) The authors concluded:

> On the basis of our extensive life-history data, we were able to calculate the total years, hours, mean hours/day, and cigarettes/day to which the subjects had been exposed to tobacco smoke at home or at work.
>
> Despite such detailed accounting, we were unable to find a significant trend in the crude or adjusted RR for these 4 lifetime measurements of passive smoking.

In a 1984 publication, Koo and colleagues reported a risk estimate for women exposed at the workplace of 0.91; this negative association was reportedly not statistically significant.[10]

Another European study including an assessment of ETS exposure in the workplace and nonsmoker lung cancer was published in 1986 (Ex. 8-192). The authors wrote that "overall the results showed no evidence of an effect of passive smoking on lung cancer incidence among lifelong nonsmokers," although they presented no odds ratios for workplace exposure. The following point estimates have been presented by one of the authors of this study in a recent book: for females, 0.63 (95% CI 0.17-2.33) and for males, 1.61 (95% CI 0.39-6.60).[12]

Shimizu, et al., reported that, in their case-control study of 90 women in Nagoya, Japan (Ex. 8-283):

> Passive smoke exposure at work was not clearly associated with female lung cancer, although the relative risk was slightly elevated (RR = 1.2).

That reported relative risk was not statistically significant.

In 1992, Stockwell and colleagues published data from a case-control study of nonsmoking women in Florida. (Ex. 8-292) The authors wrote: "We found no statistically significant increase in risk associated with exposure to environmental tobacco smoke at work." However, they failed to present the data associated with this index of exposure.

In 1985, Wu and colleagues reported on a case-control study of women in a Los Angeles, California tumor registry. (Ex. 8-326) For adenocarcinoma, the authors wrote, "we did not observe any elevated risk associated with passive smoke exposure . . . at work (RR = 1.3; 95% CI = 0.5-3.3)."

A joint Chinese-American study of women in Shenyang and Harbin, two industrial cities in northeast China, reported that 228 cases and 301 controls had been exposed to ETS in the workplace.[9] A relative risk of 1.1 (95% CI 0.9-1.6) was calculated, which the authors described as a "small excess risk," although it was not statistically significant. The authors also noted that "there were no significant dose-response trends associated with years of passive smoke exposure at work."

While the same criticisms that apply to the spousal smoking risk estimates apply to the workplace risk estimates (no actual measurements of exposure, failure to account for sources of bias and for potential confounding factors, poor study design, weak reported associations), the workplace data are, nevertheless, most appropriate for OSHA's purpose.

As illustrated in Table 1, only two of the reported risk estimates are statistically significant. The overwhelming majority, 16 of 18 risk estimates, are not statistically

significant. **Taken as a whole, these data do not support the existence of an association between workplace exposure to ETS and lung cancer risk in nonsmokers.**

> **Analyses of the workplace data as a whole, which were not considered by OSHA, do not support the claim of an increased risk of lung cancer due to ETS exposure in the workplace**

As EPA did in its Risk Assessment on ETS, to justify extrapolating from spousal smoking to other exposures, OSHA makes the claim at 59 FR 15994 that:

> [H]ealth effects observed and the risk estimates calculated from studies of the general population, or of selected subgroups, such as nonsmoking wives of smoking husbands, are relevant to the working nonsmoking population.

OSHA continues:

> [R]isk estimates based on residential exposures are expected to accurately reflect occupational risks in most workplaces and possibly underestimate the risk in some workplaces.

By neglecting the data on ETS exposures in the workplace available in the studies of spousal smoking and lung cancer, OSHA

overlooks a serious inconsistency in these data, as did EPA. As recently noted by LeVois and Layard:[13]

> The EPA's fourth point, that ETS exposure in workplaces is comparable to home exposure, and therefore if home exposure can cause lung cancer, so can workplace exposure, is not an argument for dismissing the workplace epidemiology at all. Rather, it simply avoids the question of why, if domestic and workplace exposures are comparable, the combined workplace data do not indicate any ETS-lung cancer association.

LeVois and Layard conclude:

> The fact that workplace studies produce a risk estimate that disagrees with the estimate derived from aggregated spousal smoking studies cannot be dismissed by making speculative assumptions about study design validity. . . . The workplace ETS study design avoids problems of spousal concordance with respect to lung cancer risk factors and introduces fewer potential biases and confounders than are present in the spousal study design. Thus, workplace ETS-lung cancer data are probably <u>less</u> flawed than are the spousal smoking data. [authors' emphasis]

OSHA should utilize all the available data on workplace ETS exposures from the epidemiologic studies on ETS and lung cancer

In its Notice, OSHA arbitrarily chose the workplace smoking risk estimate from the Fontham, et al., (1991) study to use in its calculation of risk. (Ex. 8-106) As detailed above, the reported result of Fontham, et al., both in the 1991 report and the 1994 report, is one of only two statistically significant risk estimates for workplace exposure. OSHA fails to address what would have happened to its calculations of risk if the risk estimate from another of the workplace studies, such as that of Janerich, et al., were used instead.

Moreover, the statistical technique of meta-analysis, although of somewhat questionable applicability to the analysis of epidemiologic studies such as these,[14] has been applied to the workplace data. Recent meta-analyses generated summary risk estimates, based on those studies then available, of 1.01 (95% CI 0.92-1.11)[13] and 0.98 (95% CI 0.89-1.08).[12] These risk estimates are not statistically distinguishable from 1.0, the "no effect" level in epidemiology. Although meta-analysis has its limitations, it does provide one means of assessing all the available data.

OSHA's calculation of attributable risk is based on a single, arbitrarily selected risk estimate; it would seem to be highly sensitive to changes in the values of the variables used; OSHA fails to discuss the uncertainty of this estimate

At Section IV.D. (59 FR 15995), the choice of the risk estimate from the Fontham, et al., 1991 study (Ex. 8-106), which OSHA attempts to justify on the basis of the quality of the study (without discussing published criticisms of Fontham, et al.[15,16]), nevertheless seems arbitrary, given the amount of additional workplace data available. OSHA arbitrarily ignores 13 other studies that give workplace risk estimates, and arbitrarily switches from reliance on the spousal smoking data in the rest of its text, to a workplace estimate for the attributable risk calculations.

The Fontham, et al., 1994 study, though based on a large sample of nonsmoking women, may be criticized for a number of shortcomings

If OSHA, as anticipated, relies on data from the second report on the Fontham study[11] to replace the data from the interim report cited in the Proposed Rule, it is submitted that OSHA should do so carefully. The makeup of this study population is likely quite dissimilar to the United States workforce as a whole. OSHA has available data from 14 worldwide workplace studies or nine

United States workplace studies, including data on both men and women. Rather than arbitrarily choosing one study, which, despite its claimed methodological advances, still does not resolve the issues of accurate exposure assessment or misclassification of smokers as nonsmokers, OSHA should consider all the available data on workplace exposures to ETS.

The following comments address the 1994 Fontham, et al., report:

- Despite the study's use of cotinine to assess current tobacco use, the authors acknowledge that misclassification of ever smokers as lifetime never smokers is "problematic" because there is "no biomarker of lifetime tobacco use." Moreover, only slightly more than half (54%) of cases had cotinine determinations. Thus, not even recent active smoking was excluded for nearly half of the cases.

- While the authors stress that their study is a multicenter case-control study, if the characteristics of the study population are examined, it is seen that the vast majority of cases and controls come from two areas in California (Los Angeles and the San Francisco Bay area). Yet, the authors do not indicate that air pollution, which has been suggested to be a lung cancer risk factor,[17] was "adjusted" for in their

analyses. In fact, the authors provide no breakdown of the data by study center, and it is not possible to ascertain whether the reported risks were consistent across the centers. Heterogeneity in the data among study sites would argue against combining the data as was done in this paper.

- Among other potential confounders that were not considered in the analyses is dietary saturated fat intake, recently reported by Alavanja, et al., to be associated with relative risks as high as 6.0 to 11.0.[18] The risk was highest in nonsmoking women with adenocarcinoma; over 75% of the cases in the Fontham, et al., study were adenocarcinomas. Alcohol consumption, another potential confounder, was also not mentioned. Recent studies have reported that smokers, and the persons living in their households, are likely to consume more fatty foods and more alcohol.[19,20]

- The presentation in the Fontham, et al., study does not provide data for the possible associations between diet and other potential confounders and lung cancer. It is thus impossible to judge whether fruit and vegetable consumption was associated with lung cancer risk but not with smoking status, or with smoking status but not with lung cancer risk. The reader must take the authors' position that the factors

were not confounders at face value, without the opportunity to examine the data.

- The "adjustment" of the reported risk estimates is difficult to interpret, as the adjustments take into account both study design variables (e.g., subject age) and potential confounders. It is not possible to determine what might be affecting the adjustment. In particular, the dramatic upward shift (1.12 to 1.39) in the risk estimate for workplace exposure is puzzling and surprising. The shift is opposite the direction expected, and is of a magnitude unlike the other adjustments in the paper.

- The study population characteristics also reveal that over 70% of cases and controls were aged 60 or older. This calls into question the accuracy of childhood exposure estimates provided by these women, who were asked to recall parental smoking habits of more than 50 years ago.

- Furthermore, the socioeconomic and educational composition of the study population argues against generalizing results reported from this study to the United States population as a whole. The majority of cases and controls were drawn from low socioeconomic strata: more than 40% had household incomes of

less than $20,000. In terms of education, one-third of cases had no more than a high school education.

- The conclusion stated in the abstract of the paper -- "Exposure to ETS during adult life increases risk of lung cancer in lifetime nonsmokers" -- gives the appearance that the reported results are applicable to both men and women, when in fact, only women were studied.

- Fontham and colleagues neglect to discuss the available workplace data from other spousal smoking studies. Of particular interest is the note that several large recent case-control studies (Janerich, et al.; Brownson, et al.; Stockwell, et al.; Wu-Williams, et al.) have reported results for workplace exposure that were <u>not</u> statistically significant. Fontham, et al., should have put their data in context.

OSHA FAILS TO REFERENCE THE NUMEROUS CRITICISMS THAT HAVE BEEN MADE CONCERNING THE EPIDEMIOLOGIC STUDIES OF REPORTED ETS EXPOSURE AND LUNG CANCER

At 59 FR 15980, OSHA writes:

> The great number of epidemiologic studies available on ETS were conducted by different researchers, on different populations, in various countries with diverse study designs. This extensive amount of data increases confidence that the associations seen between ETS exposure and the development of lung cancer is [sic] internally consistent and is [sic] not due to artifacts or a product of some unidentified, indirect factors unlikely to be common to all of the studies.

OSHA fails to reference these claims.

OSHA does not address the issue that actual ETS exposures were not measured in any of these studies reporting data on workplace exposure or spousal smoking. These studies relied upon questionnaires to provide an estimate of exposure, usually presented as the smoking habit of the cases' husbands. Concerns about the reliability of questionnaires used in ETS studies have been raised in the scientific literature.[21-23] (See also discussion of ETS exposure studies elsewhere in this submission.)

Based on the text of the Proposed Rule, OSHA does not appear to be aware that members of the scientific community have criticized the epidemiologic studies on ETS exposure and lung cancer for failing to consider certain factors, namely bias and confounding, that could affect the validity of the studies' risk estimates. The impact of such factors is particularly important in studies that, like these studies, report risk estimates that are "weak."[24] A weak association is represented by a risk estimate of less than 2.0 or perhaps even less than 3.0.[25-27] As Wynder notes[25]:

> [E]pidemiology has problems when the associations are of a low order of magnitude. In such instances, findings in the literature are, in general, inconsistent. . . .
>
> When risks are small, and especially when effects occur many years after their causes, detecting them, estimating their magnitude, and assessing their importance for the community in light of other relevant factors pose problems of study design, data collection, analysis, and interpretation which can be exceedingly difficult. (p. 139)

A detailed discussion of criticisms relevant to evaluation of the spousal smoking studies may be found in the Philip Morris submission to OSHA's Request for Information on Indoor Air. (Ex. 3-1074, Question 2(a)iii) Other submissions discussing the limitations of these studies include Exs. 3-331 and 3-1195. Additional recent articles are appended with this submission (e.g., Layard, 1992; Katzenstein, 1993).[28,29]

In the Proposed Rule, OSHA simply writes, without referencing its contentions, that:

> Many potential sources of bias, such as publication bias (the tendency of scientific journals to publish studies with positive results), misclassification bias (smokers or former smokers claiming to be nonsmokers), and recall bias (the reliance on self-reporting of personal smoking habits and exposure to others' tobacco smoke) can not account for the elevation in risks seen in these various studies.

OSHA must explain how it arrived at this conclusion, given the published literature suggesting that such sources of bias may indeed account for the reported elevations in risk. (See Ex. 3-1074) As but one example of a reference contrary to OSHA's claims, Lee has reported that a realistic smoking status misclassification rate of less than 3% is sufficient to explain the risk estimates calculated in the U.S. spousal smoking studies.[12] If OSHA has analyzed the literature on bias, such an analysis should be presented.

OSHA, like EPA, has the erroneous impression that a single confounder must necessarily apply to all the studies in order for it to be important

Despite OSHA's claim to the contrary, the very diversity of the spousal smoking studies makes it exceptionally difficult to compare them in any meaningful way. It is not logical, given this diversity, to expect that the same lifestyle variables would pertain to a population in China as to a population in Sweden or the United States. A confounder need not be common to all the studies.

In epidemiology, a confounding factor must meet one condition: it must be associated both with the exposure variable being investigated (here, spousal smoking), and with the endpoint under consideration (lung cancer). As noted in a recent review[30]:

> Because the relative risks or odds ratios for human diseases reported to be associated with ETS exposure are typically no larger than the risks for confounding lifestyle factors, epidemiological studies of the association between ETS exposure and chronic disease should be designed to maximize data quality and statistical power.
>
> The ETS and chronic disease epidemiology studies conducted to date have not adequately controlled for all of the known confounding variables.

Another 1992 review addressed the possibility that confounding factors may have a combined effect on estimations of lung cancer risk.[31]

> In the absence of calculations of lung cancer risk when multiple factors apply, one can only speculate on the combined effect on an individual who, for example, might have a family history of lung cancer (RR = 2-4), lived in an urban area (RR = 1.2-2.8), worked in an occupation associated with elevated lung cancer risk (RR = 2 or more), was among the physically less active groups of the population (RR = 2) and, if a female, had the risk associated with a short menstrual cycle (RR = 2.2).

While these factors may not truly be confounders (that is, associated both with spousal smoking and with lung cancer), but instead, independent lung cancer risk factors (associated with lung cancer but not with spousal smoking), their potential contributions to lung cancer risk have not been adequately assessed in the spousal smoking studies.

A detailed discussion of confounders is presented in Philip Morris' submission to the docket on OSHA's Request for Information. (Ex. 3-1074; Response to Question 2(a)iii)

Since the time that submission was prepared, new studies have been published that provide additional relevant data on potential confounders of the claimed ETS-lung cancer relationship,

in particular, the possible effects of diet.[18-20,32-34] Some of these are briefly reviewed below.

Recent studies indicate that the diet of nonsmokers is related to the presence or absence of smokers in the household. The studies report that nonsmokers who live in smoking households "have a diet more like smokers," consisting of more fried and fatty foods, more alcohol, and less fresh fruits and vegetables. Some authors suggest that differences in lifestyle, such as diet, may influence differences in disease risk reported when smoking and nonsmoking households are compared.

For instance, a 1992 British study examined the consumption of fried foods, fats, fruits, vegetables, and sweets in smokers, nonsmokers, and exsmokers.[19] The authors reported that nonsmokers who live in smoking households "have a diet more like smokers," and that "diet could be an important confound in epidemiological studies of ETS." The authors also noted:

> Our analysis showed that non-smokers in smoking households ate fried food more often, more chips [french fries], less fruit in winter, more butter and less margarine high in polyunsaturates than non-smokers in non-smoking households. As we have pointed out, these habits are thought to increase the probability of cancer.
>
> These results suggest that it is wise to show caution when interpreting the disease patterns

> of non-smokers in smoking households. Studies to date have failed to take into account the effect that differences in dietary and lifestyle behaviour between 'smoking' households and 'non-smoking' households may have on the incidence of cancer or heart disease.

In a 1993 paper, the same authors reported that, in addition to having higher intakes of saturated fats, never smokers living in smoking households consumed fats more often, drank more alcohol, and ate fewer root vegetables and cereal than did never smokers living in nonsmoking households.[20]

A large study conducted in the U.S. recently reported that nonsmoking women who consumed large amounts of saturated fat (i.e., a kind of fat found in meats, butter, and lard) had strongly elevated risks for lung cancer.[18] Although the authors did not compare their data on fat intake to household smoking status, they did note that "passive smoking did not affect risk estimates" in their study. The risk of lung cancer among nonsmokers who reported high consumption of saturated fats was almost three times higher than any overall risk estimate reported in the 35 spousal smoking studies. Indeed, the risks reported in the study for lower levels of estimated saturated fat consumption are comparable to any of the overall risk estimates reported in the studies on spousal smoking.

Another recent U.S. study reported that higher intake of raw fruits and vegetables, vitamin E supplements, and dietary beta carotene (a precursor of vitamin A) is associated with a statistically significant reduction in nonsmoker lung cancer risk.[32] If, as other data suggest, smokers and the persons who live with them are likely to consume less of these food items, then those individuals might have an increased risk of lung cancer. Whole milk intake was also greater among lung cancer cases in this study, which the authors suggested possibly "reflects an effect of dietary fat."

OSHA'S TREATMENT OF THE SPOUSAL SMOKING DATA IS NOT ONLY INAPPROPRIATE, BUT INACCURATE AND INCOMPLETE

Regardless of the fact that OSHA's reliance on the spousal smoking data in much of the Proposed Rule is inappropriate, OSHA should be aware that its treatment of the spousal smoking data is nonetheless inaccurate and incomplete. Inclusion of all available spousal smoking studies is important because these very studies contain the workplace data relevant to OSHA's jurisdiction.

OSHA's list of spousal smoking studies is incomplete and inaccurate; OSHA has failed to consider all available data

In addition to the 27 spousal smoking studies referenced by OSHA (Exs. 8-4; 8-35; 8-36; 8-38; 8-47; 8-52; 8-65; 8-106; 8-117; 8-118; 8-119; 8-121; 8-122 and 8-148; 8-142 and 8-143; 8-153; 8-158; 8-164; 8-171; 8-183; 8-187; 8-192; 8-283; 8-286; 8-292; 8-296; 8-300; 8-326), there are a number of spousal smoking studies that were not included in OSHA's Proposed Rule.[7-9,35-39] Copies of those studies published as of early 1992 were provided to OSHA in Philip Morris' submission on the OSHA Request for Information on Indoor Air (Ex. 3-1074; response to Question 2(a)iii).

One of the studies that OSHA failed to consider, Wu-Williams, et al., was conducted in China and is one of the largest case-control studies on this issue published to date.[9] Wu-Williams and colleagues report, for spousal smoking, a statistically significantly negative risk estimate. Another of these studies, Kabat (1990), is a preliminary report on a United States case-control study.[7] That initial report, which presented no statistically significant risk estimates, suggests that this study is controlling for a number of potential study design problems.

OSHA's citations of lung cancer studies at 59 FR 15980 are inconsistent when compared to the studies cited in Table IV-1.

(59 FR 15993) For instance, OSHA cites the important 1981 cohort study by Garfinkel (Ex. 8-118) at 59 FR 15980, but fails to include it in Table IV-1. (59 FR 15993) OSHA also cites a letter to the editor (Ex. 8-252) as one of the ETS-lung cancer epidemiologic studies (59 FR 15980), yet fails to reference the actual study.[39] A study by Kalandidi, et al., is included in Table IV-1, but is not referenced in OSHA's Exhibit 8.[8]

OSHA also "double-cites" at least one study, by treating Gillis, et al., (Ex. 8-122) and Hole, et al. (Ex. 8-148) as unique studies. These two papers are both reports from the same Scottish cohort study. By treating them as two separate studies, OSHA essentially counts the data twice.

Two of the papers cited by OSHA have been rejected in other reviews of the literature on ETS and lung cancer. The reasons for those rejections are described below.

- Katada, et al., 1988 (Ex. 8-175): The reference categories (i.e., nonexposed women) are too small to allow appropriate calculations of relative risk (because all of the cases reported ETS exposure). The U.S. EPA did not use the study in any of its analyses. (Ex. 8-311)

- Sandler, et al., 1985 (Exs. 8-275 and 8-276): The methodology and interpretation of this study has been criticized in the scientific literature (e.g., one scientist described Sandler's work as "seriously flawed").[40a-f] The data presented are of limited value, for instance, the lung cancer estimates are based on only two cases. Neither the U.S. EPA (1992) (Ex. 8-311) nor the National Academy of Sciences (1986) (Ex. 8-239) included this study in their summary analyses of ETS issues.

OSHA's argument in support of the case-control studies on spousal smoking is flawed and reveals a lack of familiarity with the available literature

At 59 FR 15980, OSHA states:

> [T]he relative risks that were estimated from prospective study data are similar to those estimated from case/control [sic] study data. Biases that may be problematic in case-control studies are not a problem in prospective studies. Since the results from both types of studies are similar it is apparent that these biases are not important in the case-control studies (e.g., misclassification bias and recall bias). This information strengthens the confidence of a causal connection.

OSHA fails to provide references to support the contention that biases are not a problem in cohort studies, nor does OSHA provide

references to justify its conclusion that biases are not operating in the case-control studies.

Misclassification of smoking status can certainly occur in a cohort study, just as in a case-control study.[41] For instance, a person may misreport his or her smoking status in the initial interview; personal smoking status may change over the course of the study; spousal smoking status may change over the course of the study. Follow-up in cohort studies must take these possibilities into account. Similarly, confounding factors associated with marriage to a smoker could operate in a cohort study as well as in a case-control study.

Moreover, while the cohort study may well be a preferred tool for epidemiologic research, OSHA has failed to recognize that the cohort studies on spousal smoking published to date have been criticized in the scientific literature. Of the four such studies available, two (Gillis/Hole, Exs. 8-122 and 8-148, and Butler, Ex. 8-47), have less than ten nonsmoker lung cancer cases each. These extremely small sample sizes reduce confidence in the risk estimates reported in those studies.

The other two studies, Hirayama (Exs. 8-142 and 8-143) and Garfinkel (Ex. 8-118), report data on larger numbers of nonsmoking women. The results of the two studies, however, are not

consistent, with Hirayama reporting a statistically significantly overall risk estimate, while Garfinkel reported no statistically significant overall risk for spousal smoking.

The Hirayama study has been heavily criticized in the scientific literature, for its mathematical errors, unconventional statistical methods, and inconsistencies in the data presented.[42-50] In particular, researchers have criticized Hirayama's techniques of age adjustment, an important consideration since age may act as a confounding factor in epidemiologic calculations.[44-49] It has been noted that Hirayama did not divide his study population into appropriate age groups, and that he also adjusted his analyses by husband's age, rather than by wife's age, contrary to common epidemiologic practice. Information available about the study also suggests that as few as 12% of the cases were histologically confirmed.[46] This means that bias could have been introduced if, among the 88% not confirmed, some died of causes other than lung cancer.

Thus, had OSHA critically examined the cohort studies of spousal smoking and lung cancer, and reviewed relevant literature critical of those studies, it would have seen that the cohort studies are not free of problems, contrary to its unsubstantiated claims.

OSHA fails to provide sufficient information to justify its classification of the spousal smoking studies in support of its quantitative risk assessment

At 59 FR 15992, OSHA states:

> As a first step in this risk assessment, OSHA critically reviewed epidemiologic studies associating exposure to ETS . . . with adverse health effects. The purpose of such a critical evaluation was to determine whether exposure to ETS is a causal factor in cancer . . . (59 FR 15992)

However, OSHA fails to provide adequate information for the reader of the proposal to evaluate how OSHA conducted this "critical evaluation" or review.

As noted earlier, OSHA's evaluation focuses on the data on spousal smoking. These are not the best available data for OSHA's purpose, as other data exist on ETS exposures in the workplace and lung cancer risk. Nevertheless, OSHA's treatment of the spousal smoking studies in this section of the Proposed Rule deserves comment.

In its discussion, OSHA continues (59 FR 15992-15993):

> OSHA evaluated studies on exposure to ETS to determine the importance and weight of each study in the overall hazard identification process. Of those, it was determined that fourteen showed a statistically strong association between exposure to ETS and lung cancer . . . Studies that were determined to be positive by OSHA's review standards met standard epidemiologic and statistical criteria to support causation. (59 FR 15992-15993)

OSHA's Proposed Rule does not describe in sufficient detail the criteria used to determine that certain of the spousal smoking studies on lung cancer were "positive," "equivocal with a positive trend," and "equivocal." Although OSHA lists the studies according to these categories in Table IV-1 (59 FR 15993), that table does not include any numerical data. It is not possible to determine whether OSHA used a single risk estimate from the study (and if so, which one), or whether OSHA somehow evaluated all the data presented in each study. Without the criteria used by OSHA, the classification gives the appearance of being arbitrary and based on unknown subjective standards. OSHA should clearly present its criteria for judging studies to be "positive" or to show "a statistically strong association."

In particular, it is not clear how OSHA can make this statement given the lack of statistical significance in the spousal smoking studies (only six report statistically significant overall relative risks), and specifically, the statistically nonsignificant

overall risk estimates included among the 14 studies in OSHA Table IV-1 that are described as "positive for an association" (59 FR 15980), "positive" (59 FR 15993), or "statistically strong" (59 FR 15992). Of those 14 studies, less than half report statistically significant overall risk estimates.

If the overall risk estimates reported in these studies are examined according to the criteria of statistical significance and the magnitude of the reported risk estimates, a different classification emerges (Table 2). Of 35 currently available studies, 11 report overall risk estimates less than or equal to 1.0; one of these is statistically significantly negative. These risk estimates are compatible with the hypothesis that there is no increased risk associated with ETS exposure, reported as spousal smoking.

Furthermore, 17 of the studies report overall risk estimates ranging from 1.0 to slightly greater than 2.0, none of which is statistically significant. Therefore, all of these risk estimates are compatible with the hypothesis of no association.

Only seven studies report overall risk estimates that are statistically significant. **In summary, then, 28 of 35 spousal smoking studies (80%) report overall risk estimates that do not support a conclusion of increased risk.**

TABLE 2: RESPONSE TO OSHA TABLE IV-1, BREAKDOWN OF SPOUSAL SMOKING STUDIES

Risk Estimate ≤ 1.0 -- Not Statistically Significant	Risk Estimate ≥ 1.0 -- Not Statistically Significant	Risk Estimate > 1.0 -- Statistically Significant
Brownson, et al., 1992	Akiba, et al., 1986	Geng, et al., 1988
Buffler, et al., 1984	Brownson, et al., 1987	Hirayama, 1984
Chan and Fung, 1982	Butler, 1988	Kalandidi, et al., 1990
Gao, et al., 1987	Correa, et al., 1983	Lam, et al., 1987
Janerich, et al., 1990	Du, et al., 1993	Lam, 1985
Kabat, 1990	Garfinkel, 1981	Trichopoulos, et al., 1983
Kabat and Wynder, 1984	Garfinkel, et al., 1985	Fontham, et al., 1994
Lee, et al., 1986	Hole, et al., 1989	
Liu, et al., 1991	Humble, et al., 1987	
Sobue, et al., 1990	Inoue and Hirayama, 1988	
Wu-Williams, et al., 1990*	Koo, et al., 1987	
	Lan, et al., 1993	
	Pershagen, et al., 1987	
	Shimizu, et al., 1988	
	Stockwell, et al., 1992	
	Svensson, et al., 1989	
	Wu, et al., 1985	

* Statistically significantly negative

It is particularly puzzling how OSHA can justify its description of the Brownson, et al., 1992 study's reported results as "positive" when that study reports an overall risk estimate of 1.0. According to epidemiologic convention, 1.0 is the "no-effect" level.

OSHA states at 59 FR 15993 that:

> Overall, on the basis of the studies reviewed, OSHA concludes that the relative risk of lung cancer in nonsmokers due to chronic exposure to ETS ranges between 1.20 and 1.50 . . .

OSHA provides the reader of the Proposed Rule with no support for this conclusion. (See subsequent discussion of meta-analysis.)

OSHA's choice of "summary analyses" is restricted to meta-analyses of spousal smoking data rather than the more appropriate workplace data

OSHA's Table IV-3 is titled "Published Risk Estimates for Lung Cancer." The numbers in the table are all summary risk estimates calculated by combining risk estimates from a number of epidemiologic studies, through a procedure called meta-analysis. This is not made clear in the Notice; in fact, only one of the risk estimates in the table is identified as based on "pooled studies."

(Ex. 8-311) Moreover, the summary risk estimates presented by OSHA are based on the spousal smoking data, rather than the data on workplace ETS exposures that have been published.

Setting aside the question of the appropriateness of using meta-analysis to combine data from epidemiologic studies,[21] if OSHA references meta-analyses, then its list should be accurate and complete. Table IV-3 is neither (e.g., a recent meta-analysis by Tweedie and Mengersen is excluded[51]).

For instance, OSHA fails to indicate the number of studies included in each meta-analysis, and whether the analyses are restricted to a certain set of studies. The meta-analyses are not completely comparable, as they incorporate different subsets of the universe of available studies. Moreover, a review of more recent meta-analyses reveals that the summary risk estimates are sensitive to the inclusion or exclusion of studies.

The example most illustrative of this is provided by the meta-analysis from the 1992 EPA Risk Assessment on ETS. (Ex. 8-311) EPA reported a summary risk estimate of 1.19 (90% CI 1.04-1.35) based on 11 spousal smoking studies conducted in the United States. In a recent paper, LeVois and Layard report[13]:

> Using the EPA's methods and assumptions, we have calculated a summary relative risk of 1.07 from a meta-analysis of 13 U.S. female spousal smoking studies, including these two recent studies [Brownson, et al., Ex. 8-36, and Stockwell, et al., Ex. 8-292]. This relative risk, with 95% confidence interval of 0.95-1.21, is not statistically significant.

Thus, the inclusion of two additional studies in the meta-analysis effectively reverses the conclusion of the EPA Risk Assessment on ETS. The summary relative risk is no longer statistically significant, and, therefore, does not support a conclusion of an association between spousal smoking and lung cancer.

OSHA'S CLAIM THAT ANIMAL INHALATION STUDIES SUPPORT THE "CARCINOGENICITY" OF ETS IS NOT SUPPORTED BY THE SCIENTIFIC LITERATURE

At 59 FR 15980, OSHA proposes: "Animal studies have shown the carcinogenicity of cigarette smoke." This assertion, which OSHA fails to reference, is directly in contrast to a recent review of the relevant literature by Rodgman, who cautions[1]:

> Classifying a substance as tumorigenic or 'carcinogenic' can be misleading. Often, these terms are overinterpreted. One must be aware of the precise meaning and limitations of the terms tumorigenicity and carcinogenicity when applied to specific compounds and must exercise considerable care in the use of these and related terms.
>
> * * *
>
> Many of these 43 MS and/or tobacco components [claimed to be carcinogens] should be excluded from the list on the basis of published data on their tumorigenicity (or lack of it) in laboratory animals at levels determined in MS, their lack of tumorigenicity in most instances on inhalation, and the equivocal evidence of their tumorigenicity in humans at levels in MS.

In this major review, Rodgman also writes:

> [I]nhalation studies from 1936 to date involving lifetime exposure of laboratory animals to whole cigarette MS have consistently failed to induce squamous cell carcinoma . . .

> The failure to produce in MS-exposed laboratory animals the tumor type reported to be associated with smoking in humans is important not only with regards to the biological properties of MS itself but also with respect to that of diluted MS delivered to the caged animals. . . .
>
> If, as Stewart and Herrold (1962) noted, these smoke-inhalation experiments more closely resembled passive smoke (or ETS) exposure than human exposure during actual smoking, then substantial evidence is available to demonstrate that exposure to 'passive smoke' (or ETS), more concentrated than that encountered in the human situation, is ineffective in induction of the tumor type supposedly associated with cigarette smoking in humans . . .

These conclusions severely undermine OSHA's contention, and make further discussion of the literature cited by OSHA of limited value. Nevertheless, OSHA's treatment of the animal studies is scientifically unacceptable; for that reason alone it deserves comment.

OSHA's discussion of animal studies suggests a failure to understand the literature, and misrepresents the conclusions of a number of papers

OSHA's discussion of experimental animal data in the Proposed Rule represents a selective, biased review of the data. The presentation of data on animal exposures is neither balanced nor accurate.

In addition to misstatements of studies' conclusions, and misrepresentations of studies' data, OSHA's discussion of animal studies is pervaded by the following errors: assuming that mainstream smoke, sidestream smoke, and ETS are equivalent enough to be used interchangeably; and failure to acknowledge the differences among subchronic and chronic inhalation experiments. Examples of the inaccuracies and misrepresentations found in this section follow.

OSHA introduces its discussion of the animal studies by saying:

> Currently, OSHA is aware of only a few experimental inhalation studies with sidestream smoke or ETS reported in the literature. A discussion of these studies follows. [emphases added]

However, OSHA's choice of studies to include shows that OSHA did not restrict its analysis to sidestream or ETS studies. For instance, the first two papers referenced deal with gas-phase smoke. Their inclusion implies that OSHA may have made the erroneous assumption that the gas phase of whole smoke is equivalent to sidestream smoke or ETS. If so, this is a distinct misconception; any type of tobacco smoke (mainstream, sidestream, or ETS) consists of both a gas and a particulate phase. If the authors of OSHA's Proposed Rule were not operating under this

misunderstanding, then they instead misrepresented the articles under discussion.

Also, OSHA claims that "data suggest that sidestream smoke may contain more carcinogenic activity per milligram of cigarette smoke condensate than does mainstream smoke," citing to Ex. 3-689D. The exhibit available to the public was not labeled in such a way that Part "D" could be identified. This made it difficult to assess the reference for OSHA's claim.

In discussing Otto and Elmenhorst's paper (Ex. 8-247), OSHA states that this research has "shown that there are carcinogenic constituents in the vapor phase of tobacco smoke." Contrary to OSHA's assertion, the authors write: "the conclusion seems justified that tumor-inducing factors must be in the particle-phase of the smoke" [emphasis added]. The authors also indicate, in the summary of the study, that their chronic exposure regimen "had no significant effect on the spontaneous tumor-rate." Thus, this study, even given OSHA's misrepresentation of its conclusions, in no way supports OSHA's claim that the "carcinogenicity" of tobacco smoke has been shown in animal experiments.

Leuchtenberger and Leuchtenberger (Ex. 8-197) used neither ETS nor sidestream smoke as a surrogate for ETS. According

to their paper, they used "whole fresh cigarette smoke" or "its gas phase alone." The Leuchtenberger article is not an chronic inhalation study of ETS or sidestream smoke, as implied in the preceding paragraph. The results cited by OSHA (pulmonary adenomas and adenocarcinomas in male mice) are but one aspect of the data reported in the study. The authors comment that those changes could be found in control animals at a later age and lower frequency. The authors note that "chronic inhalation of the gas phase of fresh cigarette smoke did not evoke bronchogenic carcinoma" [emphasis added]; they also indicate that no bronchogenic carcinomas were observed in mice exposed to whole fresh smoke. Thus, the data from this study do not support OSHA's position.

Harris, et al., (Ex. 8-135) is correctly acknowledged as a mainstream smoke study by OSHA. With regard to their reported results, the authors stated that "spontaneous" tumors appeared in their control animals during the course of their chronic study, and said that "it would no longer be accurate to refer to the inhaled smoke:air mixtures as the cause of these tumors but merely as eliciting a higher incidence." Thus, according to the authors themselves, this study's data do not support a determination that cigarette smoke causes lung cancer in experimental animals.

In Mohr and Reznik (Ex. 8-226), one of the references cited by OSHA to support the statement "Studies have also reported hyperplasia and metaplasia in the trachea and bronchi of mice exposed to cigarette smoke by the inhalation route," the variability of the results reported from smoke inhalation studies is discussed. More importantly, Mohr and Reznik write:

> [T]he majority of investigations in tobacco smoke research have been conducted by some form of inhalation technique. . . . Nevertheless, no researcher has succeeded as yet in producing a significant incidence of pulmonary tumors. (p. 347) [emphasis added]

Thus, in its selective treatment of these data, OSHA uses a lengthy review paper to support a single statement. However, OSHA fails to discuss the concluding statements made by the authors of that review. Arguably, the most important point in this review paper is what the authors noted above: that animal inhalation studies using tobacco smoke have not reported a significant increase in pulmonary tumors in exposed animals. This statement casts doubt over all of OSHA's contentions that animal inhalation experiments support the claimed carcinogenicity of ETS.

In the discussion of a paper by Davis, et al. (Ex. 8-79), which again deals with chronic exposure to vapor-phase smoke, not sidestream smoke or ETS, OSHA presents the data in such a way that

the paper's lack of a statistically significant result is effectively obscured. While OSHA's statement that: "Pulmonary squamous neoplasms were detected in female Wistar rats exposed to a 1:5 smoke-to-air mixture . . ." is not actually incorrect, it implies that the tumors were found only in exposed animals.

If one reads the conclusion of the paper cited, the following statement is found:

> The results provide convincing evidence that, under the conditions of the experiment, exposure to VP did not increase the incidence of any kind of neoplasm at any body site. (p. 467) [emphasis added]

The authors also note that cellular changes in the respiratory tract were no more frequent in exposed rats than in control rats.

While OSHA does make an accurate statement (i.e., that neoplasms were produced), its failure to present the complete picture, namely, that neoplasms were reported no more frequently among exposed than unexposed animals, is deceptive and misleading. The data presented in this paper do not support claims that animal inhalation studies show carcinogenic effects of tobacco smoke inhalation.

Some of the papers cited by OSHA, in this section which focuses on lung cancer, present data on tumors of sites other than the lung. Studies of this sort provide limited information pertinent to the issue at hand, namely, lung cancer. For instance, Dalbey, et al. (Ex. 8-77), reported a large number of benign adenomas or nasal tumors in chronically-exposed rats. Similarly, Dontenwill (Ex. 8-88) actually commented on the "very rare appearance of lung carcinomas" in his chronic inhalation study of hamsters (only 1 case).

Bernfeld, et al. (Ex. 8-30) reported on laryngeal changes, not changes to the lungs or bronchi. Moreover, the authors indicated that responses to chronic smoke inhalation varied greatly between the two hamster strains tested. If, then, two strains of the same species can exhibit markedly different responses in an experiment, by combining strains and species in the discussion and not considering the unique biology of the different animals tested, OSHA displays a overly simplistic view of biology.

In the discussion of the Auerbach, et al. (Ex. 8-19) study, in which dogs smoked through a tracheostomy, that is, a tube inserted into a hole cut into the animal's trachea (windpipe), OSHA did not even note that the Auerbach study used the tracheostomy method, as opposed to the exposure chambers used in the majority of the other studies discussed. More importantly, OSHA did not

address the inappropriateness of this route of exposure to either active smoking or ETS.

The Mohr and Reznik review (Ex. 8-226), cited by OSHA and discussed here previously, contains the following statements about the use of the tracheostomy method:

> [M]ost importantly, smoking through a tracheostomy is a highly artificial system and bears little resemblance to human experience. This is particularly the case when it is remembered that the normal dilution of smoke air is not achieved by this technique, with the result that the concentration of smoke is much higher than if taken in through the mouth.
>
> The most positive results have been obtained by means of tracheostomies in dogs. However, once again the extreme artificiality of this system prevents any real correlation of the obtained experimental results to humans. (p. 347) [emphases added]

Mohr and Reznik are discussing the relevance of data obtained in tracheostomy data to active smoking in humans. The relevance of these data to OSHA's Proposed Rule is certainly questionable, particularly given the extreme dilution of ETS once it is in room air.

Studies such as Grimmer, et al. (Ex. 8-127), discuss direct pulmonary implantation, in which a beeswax solution, containing a substance to be tested (here, condensed particles and

semivolatiles from sidestream smoke), is injected into the animal's lungs. The relevance of this experimental route of exposure in comparison to inhalation exposure is unclear. Moreover, the authors of the paper fail to present analyses of the statistical significance of their reported results. It is thus not possible to evaluate whether the paper supports OSHA's claims.

Dagle, et al., (Ex. 8-75) and Stanton, et al., (Ex. 8-289) report on the injection of a pellet of beeswax containing cigarette smoke condensate into the lungs of rats. Theoretically, this pellet would approximate chronic exposure, as substances "leach" from the pellet over time. However, as the authors of Ex. 8-289 note: "A major disadvantage is the unnatural exposure of respiratory epithelium to the carcinogen." Also, both these studies report simply on the development of this technique for bioassays.

In the subchronic study of Coggins, et al. (Ex. 8-59), aged and diluted sidestream smoke was used as a surrogate for ETS. This substance may be a more appropriate approximation of ETS than are other forms of tobacco smoke. While OSHA provides a factual summary of the conclusions of this study, they fail to note that effects (hyperplasia and inflammation) were reported only in animals exposed to particle concentrations some 100 times higher than typical real-world concentrations. Coggins, et al. (Ex. 8-60)

also report the same minor, completely reversible histopathological changes. The changes did not progress over longer periods of exposure, and once again occurred only at particle concentrations some 100-fold higher than real-world levels.

In a 14-day inhalation study, one would not expect lung tumors to develop. Thus, the relevance of the work of Coggins, et al., to OSHA's discussion of cancer is limited. Nevertheless, Coggins, et al. show only minor, reversible cellular changes following intense exposure to a surrogate for ETS.

At 59 FR 15981, OSHA cites to a book by Wynder and Hoffmann (Ex. 8-327) (the citation is unclear, particularly with respect to page numbers, as it is not a chapter or even a definable section), discussing "skin-painting" studies in which tobacco tar condensates were applied to mouse skin. In the very book cited, the authors write:

> The bioassay[s] for tobacco products on mouse epidermis have not answered questions on the problem of respiratory carcinogenesis. (p. 145)

Thus, the relevance of mentioning skin-painting studies in this context is unclear.

Reif, et al. (Ex. 8-259) reports on a "case-control" study in which owners of dogs that had died of lung cancer were asked about the dogs' exposures to ETS. Risk estimates for the dogs' developing lung cancer were then calculated. The study's authors note:

> The current study suffers from some of the same limitations found in the studies done in humans, i.e., small sample sizes, imprecise risk estimates, and difficulties in measuring exposure. (p. 238)

The "risk estimates" reported in this study were not statistically significant. Thus, these data in no way support the claim of an association between ETS exposure and lung cancer risk.

Not only do the data cited by OSHA fail to support OSHA's claims, but they represent a troubling approach to science. OSHA's Proposed Rule should be accurate and balanced: the complete data should be presented fairly, whether or not they support OSHA's position. To the reader unfamiliar with OSHA's process, misrepresenting data gives the impression that OSHA is using those data to achieve a predetermined conclusion, or that the staff responsible for the Proposed Rule did not understand the material reviewed.

OSHA's discussion of animal studies omits certain references concerning inhalation studies

Despite the fact that data from subchronic animal inhalation studies are of minimal relevance to the question of whether animal data support claims of the "carcinogenicity" of ETS, OSHA nevertheless chose to include some subchronic studies in its discussion. However, OSHA omitted several studies, which, in the interest of completeness, are described below. None of the studies report data supporting any permanent changes following subchronic exposure of animals to sidestream smoke at levels exceeding those encountered in "real-life" situations.

Haley, et al., 1987a, 1987b

Preliminary reports on this American Health Foundation study, in which hamsters were exposed to mainstream or sidestream smoke 7 days/week for 18 months, are available;[52,53] apparently, however, no final report has been published. In those reports, the authors note that smoke-exposed animals were living longer than were sham or cage control animals. No additional information was presented.

von Meyerinck, et al., 1989

Rats and hamsters of both sexes were exposed to sidestream smoke at a concentration of 4 mg/m^3 TPM and 25-30 ppm carbon monoxide for 10 hours/day, five days/week for 90 days.[54] The authors noted about their exposure system: "The levels in the exposure chamber were at least 1 and in some instances 2 orders of magnitude higher than reported for smoke-polluted rooms under real-life conditions." (Elsewhere, the authors described these conditions as "unrealistically high."[55]) One hundred animals of each species were exposed, 115 of each species were sham controls, and 100 of each species were room controls. The authors reported minor, completely reversible histopathological changes in the nasal cavity in rats only, and no alterations in any other part of the respiratory tract.

Teredesai and Pruehs, 1994

Male rats and male hamsters were nose-only exposed to fresh sidestream smoke (FSS) for seven hours/day, seven days/week for 90 days.[56] One group of 20 animals was exposed to FSS with a total particulate matter (TPM) concentration of 2 ug/L, one to FSS with TPM of 6 ug/L, and one served as a sham exposure group. Histopathological changes described as "slight" were reported in the nose and larynx of exposed rats, "mainly in the high FSS

concentration group." These changes were reversible following cessation of exposure. The authors noted that "[n]o smoke-exposure-related histopathological changes were observed in trachea and lungs."

Thus, these additional studies not referenced by OSHA fail to support claims that animal data support the purported carcinogenicity of ETS.

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